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VOLUME 17
1927

PUBLISHERS
AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILL.

Medical
Wahrs

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The star (*) preceding the page number indicates an original article in THE ARCHIVES. No author entries are made except for original articles. Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B, O and S, respectively.

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Archives of Neurology and Psychiatry

VOLUME 17 JANUARY, 1927 NUMBER 1

ERECTNESS IN MAN *

WALTER M. KRAUS, A.M., M.D.
NEW YORK

Introduction
The Factors Underlying Erectness
(*a*) Erectness as One of the Stereotyped Postures
(*b*) Postures Used by Various Animals in Progression in Relation to Human Erectness
(*c*) The Relation to Human Erectness of Sitting, Squatting, Climbing, Standing, Walking and Running
(*d*) Ontogenetic Development of Erectness in Man
(*e*) The Muscular Aspect of Erectness
(*f*) The Centers and Paths of Erectness
(*g*) Erectness as a Righting Reflex
Disorders of Physiologic Activities Other than the Postural Affecting Erectness
The Afferent Side

INTRODUCTION

The problem of the erect posture in man may be approached from many points of view. One of these considers its evolution and its dissolution; another point of view essential to an understanding of the problem considers a definition of those disorders of erectness which can in no way be considered true forms of its involution or dissolution. Man's erect posture may become impaired in many ways—by dyssynergy, dystonia and dyskinesis—but in only one way can it be dissolved and undergo involution; that is, by disorder of the mechanisms controlling posture. Thus, we have two problems, one dealing with the evolution and dissolution of erectness, the other dealing with the factors that make erectness impossible though not productive of its dissolution.

Once these points are brought out clearly, interest turns to the nature of the reflexes that make erectness possible, and these, I believe, are the righting and postural reflexes in the sense of Magnus and de Kleijn, reflexes that serve not only to move man into the normal position of

* Read at the Fifty-Second Annual Meeting of the American Neurological Association, June 2, 1926, Atlantic City, N. J.

HHW

erectness when he is not in it, but serve also to maintain it when he is in that position. This brings us to the question of localization and the place within the nervous system from which the efferent arcs spring.

Finally, I shall inquire into the reasons that underlie erectness, the afferent part of the reflex arc, and I shall speculate as to the rôle these play in determining the place of origin of the impulses for erectness.

FACTORS UNDERLYING ERECTNESS

Erectness as One of the Stereotyped Postures.—By a stereotyped posture is meant one that is characteristic of an entire species. The postural patterns of walking are included among them. Erectness is also a stereotyped posture. In contrast to such postures are habitual postures, such as a manner of sitting. These are not characteristic of the species but of individuals; that is, they are not common to all individuals of the species. All stereotyped postures are infracortically produced, while habitual postures are cortical in origin.

Complete erectness occurs only in modern man. It consists of a definite relation of trunk to hind limbs. They lie in the same plane and axis, cephalocaudad. The angle made between the trunk and the hind legs is 180 degrees.

In order that this stereotyped posture may exist, a number of other more essential postures are necessary. If the individual is walking, it is necessary that a number of postures integrated within the spinal cord be active. I have already described these.[1] The postural mechanisms are those concerned with flexion and extension of the axial muscles which control the trunk as well as the postures concerned in the four phases of gait.

It is also essential that the posture mechanisms controlling the position of the head should be intact. The head must be held erectly and in the midline. The additional postural mechanism that is required for complete erectness is one that maintains the trunk at 180 degrees with the hind legs. When erectness is dissolved, this angle becomes diminished and then there is an associated forward flexion of the trunk and of the head. This is the only change from the normal erect posture that can be defined as dissolving it. A consideration of this dissolution of erectness from a clinical point of view has already been discussed in a previous paper.[2]

1. Kraus, W. M.: The Difference Between a Muscular and a Neuromuscular Interpretation of Walking, Arch. Neurol. & Psychiat. **9**:184 (Feb.) 1923; Rôle d'intégration joué par la moelle dans la determination des attitudes du tronc et des membres, Rev. neurol. **2**:289 (Oct.) 1923. The Constant Relation Between Postures of Rigid and Motile States, Arch. Neurol. & Psychiat. **15**:597 (May) 1926.

2. Kraus, W. M.: An Interpretation of the Parkinsonian Syndrome in Terms of the Neuromuscular Mechanism, New York State J. Med. **22**:399 (Sept.) 1922.

Postures Used by Various Animals in Progression in Relation to Human Erectness.—The erectness that has appeared in birds, certain mammals and apes is only a fraction of that of man. The evolution of erectness is not along the same pathway as that leading to it in man. Just what the precise line of development to man has been is still undecided. However, it can be said that the stooping posture attributed to primitive man is a precursor of our erect posture, and some light may be thrown on that which preceded ours by a study of its involution. It cannot be said, however, that a pathologic manifestation in man is an equivalent of a normal manifestation of more primitive types.

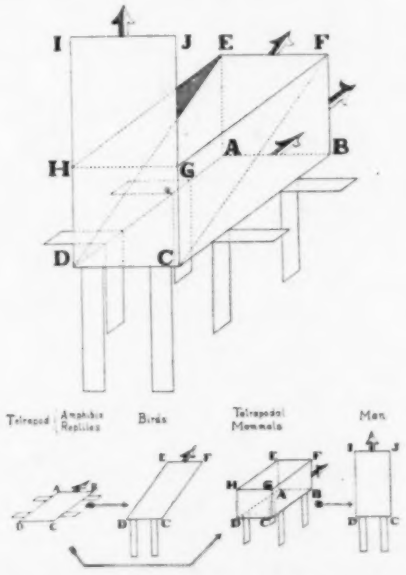

Fig. 1.—The evolution of erectness, showing the planes of the torso and of the limbs of Amphibia, tetrapodal reptiles, birds, tetrapodal mammals and man when standing. The arrow indicates the head. The four small lower figures should be examined first. The plane of bodies of Amphibia and tetrapodal reptiles is horizontal. From reptiles birds developed, and it can be seen that the legs are vertical while the plane of the torso is inclined. Along another line of development from reptiles tetrapodal mammals appeared and from them man.

It may be seen that the plane of the torso in mammals is at right angles to that of the hind legs, while in man this angle is 180 degrees. Birds, though erect, do not show this 180 degree angle of torso with hind limb.

The large figure above shows the four lower smaller figures in composite form with corresponding lettering.

The steps leading to erectness are well defined. Limbs that counteract gravity do not exist in fish. Four-limbed *Amphibia* have limbs that counteract gravity. They are the first terrestrial vertebrates. The limbs lie in the horizontal plane of the animal (fig. 1). Gradually, the

development into tetrapodal reptiles occurred, and the limbs lay more in a plane tilted downward from the midline of the trunk. Furthermore, the limbs began to be used not only for support during progression, that is, walking and running, but also for support while at rest, an important additional use. Static tetrapodal supporting, i. e., standing, preceded motile tetrapodal supporting, running, for in order for the limbs to support during walking an initial stage of standing must occur. This relation of support to standing and walking is also found in the erect apes and man. Some apes can stand erect but have not acquired the ability to walk or run in an erect position.

At the reptile stage radiation begins, which on one branch leads to erect dinosaurs and birds, and on another to mammals. The erect dinosaurs are interesting mainly in that they formed the erect ancestors of the birds, a branch that has led no further. Birds are capable of maintaining erectness far better and longer than man. One has only to think of the stork and the pelican standing for long periods on one leg. However, at least one characteristic of human erectness fails; the hind leg is not in the cephalocaudal axis, but at an angle to it (fig. 1). The devotion of the forelimbs to flying does not produce erectness as it occurs in man. It merely limits standing to the hind limbs. The mammals also develop erectness; one thinks of the kangaroo, the squirrel, the primitive primates, as *Tarsius,* of trained animals at the circus and many others; but the erectness differs from that of man; the thigh is not in line with the vertebral column, but at an angle with it. It is squatting, a kind of sitting. Even in birds, the same angle appears (fig. 1). In *Anthropoidea,* erectness occurs in gibbon, gorilla and chimpanzee, but again the posture of complete erectness, the straight line of the trunk and legs, is not present.

Certain changes occur when erectness makes its appearance. The forelimbs are released for other purposes either completely or partially; in birds, completely; in lower mammals, partially; in monkeys, partially, for the purpose of grasping the branches of trees and food. They still may use the forelimb for progression, but in most of the higher monkeys it is exclusively for arboreal progression. In man the forelimb is released and is used for purposes having only a few relations to walking. The associated swing in walking and running shows the mark of a former usage.

The leg in tetrapodal mammals has already rotated inward toward the median line and has taken its place beneath the trunk instead of at its side as in *Amphibia* and reptiles. In erect primates and man, the angle of the leg and trunk has opened; in man it has opened completely (fig. 1). In primates the cervical flexure of the vertebral column is absent; in man it enables the head to lie in the vertical axis. In infants the lumbar flexure is slight and the erector spinae small. In adults, this

flexure is conspicuous and the erector powerful. The two flexures, cervical and lumbar, disappear when erectness in man begins to be dissolved.

The Relation to Human Erectness of Sitting, Squatting, Climbing, Standing, Walking and Running.—There are still other ways of tracing the steps to erect posture, one by comparing those of the apes with those of man and the other by comparing the various postures through infancy with the age of walking and running. Man and the apes have a common ancestor. "This does not mean that man arose from any known ape, or that any ape could ever in the course of evolution give rise to a man, but that man and the ape had at some not very remote time, geologically speaking, a common ancestor. It is, however, highly probable that were we to see this common progenitor in the flesh we would be at a loss for a descriptive term to apply to it if we excluded the word 'ape.'" [3]

As has already been stated, moving about on all fours and particularly crawling are the first means of progression in terrestrial animals. The crawling of *Amphibia* becomes tetrapodal walking in higher animals. The next posture is squatting. The next, sitting, which all of the apes can do, and which preceded brachiation, that is, the use of the arms for arboreal progression, climbing. It involves a kind of erectness and may be the cause of its development in man, but as yet nothing approaching the human type is found. The next stage is standing erect, which gibbon, chimpanzee and gorilla can do, but the orang-utan cannot. Running erectly (bipodally) is a still further development which only gibbon and gorilla can do. Standing is static erectness and running motile erectness. Static bipodal support or standing precedes motile bipodal support or running as occurs in tetrapodal progression.

Running is much more difficult than standing, for running consists of a continuous repetition of falling and recovery. The movement forward on the leg of support in walking or running would result in a fall if it were not that the leg passing through flexion comes forward to prevent this. The righting reflex for erectness is continually reinvoked in the process of running. In the process of standing, it is continuously invoked.

Ontogenetic Development of Erectness in Man.—The second way of tracing the steps leading to complete erectness is by recalling what happened to man. An infant can progress through water, if suspended, as in the tub. Next it sits and then crawls, axial extension preceding appendicular as a righting reflex. Up to this point, no attempt to put the legs in line with the trunk is possible. Grasping here and there, the child next attempts to stand. Then it stands and finally walks and runs. The manner of rising to the standing position changes during the first

3. Lull, R. S.: Organic Evolution, New York, The MacMillan Co., 1925.

five years of life.[4] Before this change the child first arrives at the sitting position by a process of rotation. After 5, and in adults, the rise into the sitting position from the recumbent is directly forward.

An infant fails to walk and run, not because he cannot progress, but because he cannot stand. Walking is more complicated than standing and is the last to be learned. What is lacking when standing, walking and running are impossible is the ability to maintain the trunk and legs in line, the ability to assume erectness and, further, the inability when put into the erect posture of invoking the righting reflex which maintains it.

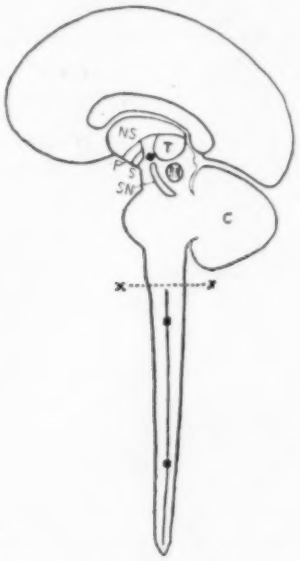

Fig. 2.—The centers of posture determination. *NS*, neostriatum; *PS*, paleostriatum; *T*, thalamus; *RN*, red nucleus; *SN*, substantia nigra; *C*, cerebellum; *X-X*, division between the brain and spinal cord. In the spinal cord, the heavy line with two squares represents the location of the centers which determine spinal postures, so-called decerebrate postures; the squares indicate the special centers for arm and leg postures. The square above the midbrain represents the approximate location of the supramesencephalic arm center and the approximate location of the center of erectness in man.

The Muscular Aspect of Erectness.—The muscles that must be in action to maintain the trunk fully extended are the axial group. Furthermore, the relative tension of the dorsal and ventral or the episomatic and hyposomatic groups must be such as to enable the erectness to exist.

It is also true that the erector spinae is most powerfully developed in man and is the muscle of human erectness. This shows that for erectness the dorsal musculature is required to act more predominantly in man than in lower animals. When it is desired consciously to increase

4. Schaltenbrand, Georges: Normal Bewegungs und Lagereaktionen bei Kindern, Deutsche Ztschr. f. Nervenh. **87**:23, 1925.

erectness, the dorsal axial muscles are used. As soon as the balance between dorsal and ventral muscles is impaired so that the ventral are relatively more active, the erectness begins to be dissolved. The postural reflex acts less well. I have already emphasized this relation between ventral and dorsal muscles. It indicates that the posture center for erectness controls in a definite way the activities of the two divisions of the axial musculature.

The Centers and Paths of Erectness.—The question now arises as to what level or parts of the nervous system are concerned with the production of the posture of human erectness. The postures underlying it have already been defined, and it has been stated that the mechanism for the postural patterns of legs and trunk in normal walking lies within the spinal cord (fig. 2).

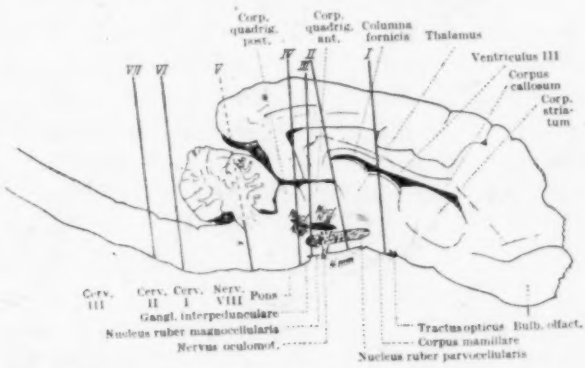

Fig. 3.—Righting reflexes in the cat. The pathways for righting reflexes extend cephalad to line II; that is, portions of the brain anterior to it may be destroyed without destroying any of them (Magnus' Körperstellung).

In a recent paper, I have emphasized that the mechanism responsible for the position of the arm in diseases such as hemiplegia and parkinsonism lies above the level of the red nucleus. Since lesions at this level release the postures of the decerebrate state which, as I pointed out with Rabiner,[5] are spinal postures analogous to those of tetrapodal walking, it would seem certain that the mechanism responsible for erectness (a more highly developed form of posture) must lie above this level. The capacity to be erect followed, from an evolutionary point of view, that of prehension in the upper extremities; since the mechanism underlying prehension lies above the red nucleus, one would look on this as further evidence that erectness must also be produced above it and somewhat above the site of production of prehension itself.

5. Kraus, W. M., and Rabiner, A. M.: On the Production of Neuromuscular Patterns by Release of Spinal Integration after Decerebration, J. Neurol. & Psychopath. **3**:209 (Nov.) 1922.

Erectness as a Righting Reflex.—The evidence of the work of Magnus [6] and his collaborators paved the way to the description by Wechsler and Brock [7] of erectness as a righting reflex. This is invoked whenever standing, walking or running are undertaken. A consideration of the levels below which Magnus and his collaborators found that righting reflexes disappeared is illustrated by figure 3. The righting reflexes which he has described of course do not include the human erectness reflex, but it is apparent that this reflex would lie at a level between the lines II and III and even above II in man.

DISORDERS OF PHYSIOLOGIC ACTIVITIES OTHER THAN THE POSTURAL AFFECTING ERECTNESS

For the proper carrying out of this righting reflex underlying erectness, a number of physiologic activities of the nervous system are required. I should like to reemphasize what I have previously stated; namely, that the various physiologic activities underlying motility are separate and distinct, one from another, both as to their centers and paths and as to their physiologic characteristics. Posture, tone, synergy and kinesis have their own characteristics and their own centers and paths. They, when normally combined, insure normal posture and movement.

A normal righting reflex is evoked by kinetic impulses acting on an intact posture mechanism in the presence of normal tone and synergy. A normal righting reflex of human erectness demands the same elements. Erectness is dissolved and involuted when the posture mechanism is diseased. Erectness is impossible or impaired though in an entirely different way when kinetic, tonic and synergic mechanisms are injured. Clinically, disorders of tone, kinesis and synergy each produce different pictures of disturbed erectness from disorders of posture It is self-evident that the disorder of erectness due to rigidity or to poor equilibrium is in no sense equivalent to the primary disorder of posture such as is seen in old age and parkinsonism. As a parallel, the use of voluntary power may in itself be intact but may be vitiated by disorders of tone, synergy and posture. Tone may be normal, but its effects may be vitiated by disorders of synergy, posture or kinesis, and synergy may be normal but vitiated by disorders of posture, kinesis or tone. It is also true that two or three of these physiologic activities may be disordered, vitiating the normal action of the others.

It is evident that dyssynergy in itself does not dissolve erectness. It does not interfere with that mechanism of the nervous system which

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6. Magnus, R.: Körperstellung, Berlin, Julius Springer, 1924, p. 753.

7. Wechsler, I. S., and Brock, S.: The Loss of the Righting Reflex (Stellreflex) in Paralysis Agitans, Arch. Neurol. & Psychiat., in this issue.

is responsible for the production and maintenance of erectness as a posture. It merely makes this maintenance impossible. A tabetic patient or one with a cerebellar dyssynergy who requires a broad base in order to stand does not show any dissolution of erectness, a return to a more primitive posture. He merely shows inability to use his muscles properly to carry out the postural reflex that underlies it.

Recently, Rademaker,[8] in a presentation of his work on the red nucleus and of the literature pertaining to this structure, has expressed the opinion that the red nucleus is the center for erectness merely because interference with the paths from the cerebellum to this structure or with this structure itself has made erectness impossible. It is, however, clear in reading his observations of reported cases of lesions in the region of this structure that what he has been dealing with is not a dissolution of the erect posture but a disorder of synergy which has made this posture impossible. If the pathway from the cerebellum by way of the superior peduncle to the red nucleus is disordered, it is certain that the normal synergic action of the cerebellum will be interfered with, and so complicated a righting reflex as that leading to erectness will also be impaired. Therefore, I cannot share the opinion that the red nucleus is in any way the primary center for the production of the posture of erectness. I cannot identify disorders of equilibrium and of synergy with those of posture.

There seems to be no question that the reflex arc of tone which originates in the muscle and ends therein is controlled in the sense of inhibition by the red nucleus.[8] If one would argue from this that the red nucleus is a center for erectness because a lesion of this nucleus permits increased tone and thus makes normal erect posture impossible, the error would again be made of identifying structures having to do with tone with those having to do with posture. If the tone is sufficiently increased, normal posture becomes impossible, but it is no more true when an increase or even decrease of tone occurs that the postural mechanism of erectness is diseased than it is true that a disorder of synergy destroys this mechanism.

If the kinetic innervation that is necessary to activate erectness is destroyed, as in flaccidity, erectness is impossible; but if the flaccidity disappears, erectness may again be resumed. On the other hand, when convulsions occur, erectness is impossible; but as soon as the convulsions disappear, erectness again occurs, as in the epileptic patient. The lesion that underlies a disorder of kinesis has not in the least disturbed the mechanism for erectness. It has merely prevented it from acting.

8. Rademaker, G. G. J.: Die Bedeutung der roten Kerne und des übrigen Mittelhirns für Muskeltonus, Körperstellung und Labyrinthreflexe, Berlin, Julius Springer, 1926.

From all of this I wish to reemphasize what I regard as the most important principle that will clarify the knowledge of motility; namely, that posture, tone, synergy and kinesis can never be identified with one another in their effects or in their mechanisms.

Many schemas have been devised to illustrate the various aspects of motility in their anatomic relation, but I believe that all of them must be "taken with a grain of salt" until such time as a clear differentiation of the four physiologic activities has been defined with much more clearness than it has been previously. It is a complex problem, but perhaps as it is realized that every part of the nervous system has its function, one may more easily become convinced that it does not share a half dozen functions with some connected structure The connections between these functional systems are for the purpose of linking and integrating different centers, and consequently different functions, for the carrying out of normal motility.

THE AFFERENT SIDE

Up to the present time, only the factors that throw some light on the motor control of erectness have been considered. By far the most important part, in my opinion, is still untouched; that is, the afferent side. The idea of neurobiotaxis has not been applied extensively to the problems of human neurophysiology to determine the reasons for the location of various centers in certain parts of the brain. According to this law, motor centers are laid down in close relation to the sensory centers and paths that affect them. It is reasonable to believe that the motor centers for erectness are not far from the sensory centers which carried up the impulses that occasioned their appearance.

The center for extension of the leg does not lie in the spinal cord in monkeys and man as in lower animals, but has been attracted upward into the brain stem. This posture is one of those underlying erectness. It is of interest that in birds, and I refer particularly to chickens, this center still seems to lie within the spinal cord although the animal is erect. It has not been attracted upward. If a chicken's head is cut off, he still continues to run about for a short time.

The question is, What has attracted the center for erectness in man to a level above that of the red nucleus? Near which new sensory station has it appeared? Indeed, what new sensory station has appeared that would demand erectness as a concomitant manifestation? One thinks, of course, of the emancipated arm and hand and of binocular vision, of stereognosis of touch and sight.

The sense of stereognosis in the hand has a representation in the thalamus, and it is not far from this region that I have placed the level for the prehensile arm. The powers of stereognosis and prehension seem to have increased together.

The sense of sight and binocular vision has gradually increased in complexity and refinement by the appearance of central vision, convergence and accommodation. It is apparent, I think, that an animal on all fours can turn less quickly for fixation of vision than one standing on his hind legs. It seems, therefore, that one reason for being erect is to allow the recent and delicate functions of the eyes to be carried out more efficiently.

LOSS OF THE RIGHTING REFLEX IN MAN

WITH SPECIAL REFERENCE TO PARALYSIS AGITANS *

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AND

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In studying paralysis agitans we have been more and more impressed with the fact that its essential features are disturbances of posture and defects in the performance of "associated and automatic" movements. We believe that these represent losses of the righting reflexes (Magnus-de Kleijn) in man. We are further of the opinion that the poverty of emotional expression seen in this disease can also be correlated with impairment of higher righting reflexes.

In analyzing paralysis agitans, it becomes evident that tremor may be absent, that paralysis of voluntary movement is wanting and that even rigidity may occasionally be missing. But in no case does one fail to observe the characteristic postural disturbance together with poverty or loss of automatic and associated movements. The essential feature of the postural disturbance is a reversion, more or less, to the quadripedal stance of the lower mammal, or as Kraus [1] states, "a dissolution of erectness." Another important element is the inability to right oneself quickly, to readjust one's center of gravity, as shown by festination and the various pulsions. In other words, if the center of gravity is displaced forward, the normal man automatically rights himself; the same adjustment occurs on backward and lateral displacements. In paralysis agitans, in which this righting ability is impaired, the person cannt make such equilibratory adjustment, and hence persists in moving in the direction imposed by the shifting center of gravity. We would emphasize that the characteristic posture and the various pulsions of paralysis agitans are the static and motile phases, respectively, of the same underlying disturbance. A study of the righting reflexes as described by Magnus and de Kleijn reveals a number of comparisons, which permit the inference that in paralysis agitans we are dealing with a loss of a fundamental righting mechanism in man.

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* Read at the Fifty-Second Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 1, 1926.

* From the Neurological Service, the Montefiore Hospital.

1. Kraus, W. M.: Le phénomène de Holt: les effets, pendant la marche, du déplacement en avant du centre de gravité du corps sur la position maxima de support de la jambe; sa signification à l'état normal et dans les conditions pathologiques, Ext. de An. d. méd. **15**:1 (Jan.) 1924.

OBSERVATIONS OF MAGNUS

It will be recalled that Magnus [2] has described various reflexes in animals in which the brain stem has been transected at certain levels. In the decerebrate animal, he speaks of the reflexes concerned in standing as "Stehreflexe," stressing the tonic neck and tonic labyrinthine influences. When the transection is made at or posterior to a level located between the anterior and posterior quadrigeminal bodies, the animal is incapable of righting itself; it cannot resume its normal posture from any abnormal position in which it is put. Further, the extensor muscles are everywhere hypertonic. When, however, the transection spares the mesencephalon, leaving it in functional continuity with the lower parts of the neuraxis, the animal possesses the means for righting itself ("Stellreflexe"). In addition there is normal distribution of tonus. Such a midbrain animal possesses a mechanism for the reception and distribution of stimuli concerned in righting. The righting reflexes are in constant activity to maintain the animal in its optimum posture whenever, as is constantly happening, there is a departure from it. The stimuli activating the reflexes come from: (1) the labyrinths, (2) the body surface acting on the head, (3) the body surface acting on the body, (4) the neck-axial muscles and (5) the eyes.

It may not be amiss at this point to illustrate the various mechanisms as Magnus describes them. If a monkey whose thalami have been transected is suspended in the air, head down, its head becomes dorsiflexed. The stimulus producing this reaction arises in the labyrinths, because if they are excised the head hangs downward in response to gravity. This labyrinthine function operates alone only when the animal is suspended in space. If such a "thalamus," labyrinthless animal is laid on its side on a flat surface, its head will again right itself and resume its normal position. The stimuli activating the center now arise from the asymmetrical pressure on the underlying body surface. To prove this, Magnus applies a lightly weighted board to the upper side of the animal and neutralizes the stimuli coming from the underlying surface; the head of the animal no longer rights itself. If he removes the upper board, the head again resumes its normal posture. This illustrates the effect of the body-righting reflexes on the head. The body-righting reflexes influencing body posture are also important. If the eyes of a labyrinthless ape are covered and it is suspended in the air, it loses all orientation in space. If it is held head down and its forepaws touch the floor, the loosely dangling head becomes dorsiflexed; moreover, the sightless ape is able to climb about its cage with normal dexterity, thanks to these axio-appendicular stimuli acting on the body.

The neck-axial chain of reflexes is of especial interest. As soon as the head rights itself the neck musculature transmits stimuli to the body,

2. Magnus, R.: Körperstellung, Berlin, Julius Springer, 1924, p. 753.

bringing the latter into normal alinement. Thus, a chain of reflex activity starts with the labyrinths or body surface reflexes acting on the head, the head activating the neck musculature, and the latter the axial musculature in a caudal direction.

In apes and in man, the eyes are important organs for righting. After losing its ability to right its head following bilateral labyrinthectomy, an ape gradually (within a fortnight) compensates for this loss by the use of its eyes. Postural orientation is achieved by fixing its gaze on objects. These are the optic righting reflexes.

Thus the differentiation is largely based on the variety of the afferent pathways used, and to a certain extent on the localization of different centers as revealed by animal experimentation. Without citing at too great length from Magnus' monograph "Körperstellung," we would emphasize that the midbrain and interbrain regions contain the "centers" concerned in these reflex postural adjustments.

OBSERVATIONS OF OTHER AUTHORS

In the past decade, the familiar studies of the Vogts, J. R. Hunt, Lewy, Strümpel and Foerster have added considerably to our knowledge of paralysis agitans. Hunt's emphasis on the loss of associated and automatic movements and the pathologic changes of the efferent pallidal system marked a real advance in the physiologic and pathologic conception of the disease. In a recent analysis, Foerster [3] stresses the following: tremor; increased plastic tonus with increased resistance to passive stretching of muscles; fixation of the muscle to whatever length is imposed on it; tonic prolongation of the response to electrical and reflex stimulation; absence of spread of reflex movement; loss of postural reactions; absence of movements of emotional expression or their tonic prolongation when present; limitation of range, absence of spontaneity, slowness in initiation, performance and cessation of all voluntary movements; absence of normal associated movements and of signs referable to pyramidal tract involvement. Essentially, Foerster emphasizes the rigidity and the loss of associated automatic postural adjustments with their emotional components.

Walshe [4] stresses the part played by the rigidity, saying that "the rigidity, if not the sole factor in the causation of the characteristic slowness of intonation and performance and the restriction of range of voluntary movements in paralysis agitans, is certainly a very potent factor."

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3. Foerster, O.: Zur Analyse und Pathophysiologie der striären Bewegungstörungen, Ztschr. f. d. ges. Neurol. u. Psychiat. **73**:1, 1921.

4. Walshe, F. M. R.: Observations on the Nature of the Muscular Rigidity of Paralysis Agitans and on Its Relationship to Tremor, Brain **47**:159 (May) 1924.

We are not directly concerned here with the complicated question of rigidity or tonus, but we cannot subscribe to the notion that rigidity conditions the postural disturbances in this disorder, as implied by Walshe. We have observed the typical posture and defective associated and automatic movements of paralysis agitans in a youth with post-encephalitic symptoms, in whom there was no rigidity. Moreover, in the cat whose mesencephalon is transected between the anterior and posterior quadrigeminal bodies, the decerebrate (extensor) hypertonus does not interfere with the neck-axial righting reflexes (Magnus).

Foerster and Lewy [5] have noted the loss of certain "reactive movements" of flight, fright, defense, surprise, etc. In terms of our newer physiologic concept, these reactions are reflex righting maneuvers.

Movements of emotional expression are notably lacking in paralysis agitans. We believe that, with man's assumption of the upright posture and his evolved capacity to give motor facial expression to his emotions, the facial mimetic motor patterns became fused with the righting reflexes. In man the righting reflexes have acquired an affective component.

The centers for the adjusting mechanism described by the Dutch investigators are placed in the midbrain. A special type of reflex ("Sprungbereitschaft"), which is the posture assumed by an animal as it lands at the end of a jump, has a center located higher in the diencephalon. Rademaker [6] performed an ingenious experiment in which he severed the decussation of Forel (rubrospinal tracts) in a rabbit transected at the thalami, and thereby abolished the animal's ability to right itself. This would seem to demonstrate that the red nuclei constitute one of the important centers for the reflexes of righting in cats and rabbits. Aside from the midbrain centers governing righting, there is evidence that other higher regions subserve similar functions. For instance, the visual cortex is concerned in the optic righting reflex. Perhaps there are olfactory righting reflexes in certain animals (dogs), as yet undefined, operating through subcortical areas. If the emotional reactions ("Reactivbewegungen") are construed as partial righting reflexes, there must be additional cortical or subcortical localizations for this function.

In the cases of paralysis agitans in which the pathologic change is in the substantia nigra, the question presents itself as to whether the

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5. Lewy, F. H.: Die Lehre von Tonus und der Bewegung, Berlin, Julius Springer, 1923, p. 673.

6. Rademaker, G. G. J.: The Significance of the Red Nuclei and the Other Parts of the Mesencephalon for Muscle Tonus, for Normal Attitudes and for Labyrinthine Reflexes, Leyden, Eduard Ijdo, 1924; abst. trans. by Walshe, F. M. R.: Brain **47**:390, 1924.

destruction of this mesencephalic structure may not possibly be responsible for the loss of some of the righting reflexes. In those cases of paralysis agitans showing pallidal (diencephalic) pathologic change, a higher center must be postulated. We are ignorant of the afferent pathways concerned. We have blindfolded patients with this disease and have elicited propulsion or noted exaggeration of the pulsions in some cases, and have failed to influence others. When patients with paralysis agitans were suddenly overthrown, it was observed that they dropped without being able to break the fall. How much this was due to rigidity and how much to the loss of righting was obviously impossible to determine. Man does not lend himself very well to such investigations because of the rich, intricate conditioned reflexes introduced by the cerebral cortex at the psychic level.

Certain observations found in the literature show that others have described signs referable to the midbrain and interbrain which, we would point out, are disturbances in reflex righting. Souques [7] described a sign in cases of paralysis agitans which he deemed of considerable diagnostic significance. This consists of suddenly tilting backward the chair in which the patient is seated while he is unaware of the nature of the maneuver. Normally, there is sharp extension of both lower extremities at the knee joints. In paralysis agitans the knees remain flexed. We interpret the normal reaction as an attempt to restore erect posture. The Souques' phenomenon, then, may be regarded as a lost righting reflex. The question still remains as to whether this particular righting adjustment is mediated by the labyrinths or a higher mechanism.

Von Sarbo [8] described a sign, which he called "hyptokinesis," consisting of retropulsion on strong dorsiflexion of the head. In some cases this maneuver alone sufficed to induce the backward swaying; in others it was necessary also to close the eyes before the sign could be elicited. Von Sarbo felt that the manifestation was due to a lesion in the nucleus ruber system. Analysis of its mechanism in terms of the righting reflexes clarifies the otherwise obscure phenomenon. The tilting of the head backward displaces the center of gravity enough to produce a loss of righting in certain cases. Shutting the eyes eliminates the important optic righting reflexes and either intensifies or elicits the loss of righting.

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7. Souques, M. A.: Rapport sur les syndromes parkinsoniens, Rev. neurol. **36**:555 (June) 1921.

8. Von Sarbo, A.: "Hyptokinesis" und "rubral Ataxie" als Symptom der Gehirngeschwülste der mittleren Schädelgrube speciell des Mittelhirns: Versuch das "rote Kern System" als gleichgewicht Zentrum aufzufassen, Klin. Wchnschr. **1**:1597 (Aug.) 1922.

CONCLUSIONS

The similarity of the von Sarbo maneuver to the Romberg sign suggests that the latter also comes within the realm of the optic righting reflex. The question may be asked: How much does the fundamental optic (visuocerebral) righting reflex assist the spinothalamocerebral pathway concerned in coordination? The optic righting reflexes normally assist in the maintenance of that coordination which is based on stimuli transmitted by spinothalamocortical pathways. The exclusion of vision in the presence of that type of sensory ataxia results in the Romberg sign. The absence of the latter in purely cerebellar types of incoordination is due to the comparatively minimal influenecs of the optic righting reflex.

Although much of this work is speculative, we feel that it is a justifiable attempt to correlate recent physiologic experimental work with everyday clinical neurology. More observation and investigation are necessary to give the exact anatomic and physiologic basis for the contention that the essential postural disturbance in paralysis agitans is a defect of the higher righting mechanisms. Dissolution of erectness, loss of the reaction movements, and loss of associated and automatic movements may thus be reinterpreted in the physiologic terms of the "righting reflexes" in man.

NOTE.—After this paper was finished, we were privileged to have Professor Magnus' comment on it while he was in this country in June, 1926. With us, he feels that the subject is still in a speculative phase and that our paper is a justifiable "Fragestellung." He correctly points out that his animal experiments have disclosed no righting reflexes of the kind described in this paper. For the present he suggests using the phrase "reflexes opposing bodily displacement" for the disturbance in postural adjustments detailed above, in order to delimit them from his righting reflexes, which have such an accurate anatomicophysiologic basis, because of his painstaking researches. In any event, "the reflexes opposing bodily displacement" in a broad sense act in a righting capacity. What is most seriously needed (as Professor Magnus pointed out) is a careful analysis and definition of these reflexes on a physiologic and anatomic groundwork.

STUDIES IN DECEREBRATION

IV. INTEGRATED REFLEXES OF THE BRAIN STEM *

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In two previously published communications [1] it was pointed out that simultaneous ligation of the basilar and carotid arteries produced a decerebrate preparation which was free from the objections of mutilation and shock. In an article just published we [2] have described the results of destruction of the labyrinths in animals decerebrated by the anemic method.

From this study it appeared that the labyrinths exert a strong influence on the extensor reflex of the neck, which tends to produce a fixed position of the head in extension, with a consequent marked extensor rigidity in the forelegs. Not only does this position evoke an extensor rigidity in the forelegs, but the play of labyrinthine tone on the extensors of the neck is sufficient alone to reinforce the extensors of the forelegs when the head is passively flexed in an ordinary decerebrate animal. When the labyrinths are destroyed and the head is suddenly released, marked and sudden flexion occurs as the result of gravity, and the forelegs are flexed and adducted. The rigidity in flexion is as great as is the rigidity in extension in the ordinary decerebrate animal. The position of the forelegs in flexion is retained indefinitely, until the head is passively extended, or some phasic reflexes elicited.

It was evident from this study that at least two patterns of tonic postural reflexes could be elicited from the brain stem functioning in an ordinary decerebrate animal. We were now concerned with the possibility of releasing other reflexes, and determining what factors inhibited the predominance of labyrinthine tone when higher levels of the brain stem remained intact. In the course of some unpublished work on epilepsy it was necessary to isolate a segment of the basilar artery by

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* From the Laboratory of Surgical Research, Northwestern University Medical School.

* Read at the Fifty-Second Annual Meeting of the American Neurological Association, at Atlantic City, N. J., June, 1926.

1. Pollock, L. J., and Davis, Loyal: A Method of Decerebration, Arch. Neurol. & Psychiat. **10**:391 (Oct.) 1923; An Acute Decerebrate Preparation, ibid. **12**:288 (Sept.) 1924.

2. Davis, Loyal, and Pollock, L. J.: Studies in Decerebration: III. The Labyrinth, Arch. Neurol. & Psychiat. **16**:555 (Nov.) 1926.

ligatures some distance apart. During the same work animals were decerebrated by a ligation of the basilar artery much higher than in our ordinary animals. The preparations resulting from both of these procedures showed great activity. The experiments were therefore repeated. Two groups of animals were prepared, the one in which only a double ligature of the basilar artery was made, and the other in which a ligation of the basilar artery was made just posterior to the pituitary fossa and the carotid arteries were then tied. Curiously, the resulting preparations resembled each other closely.

An abstract of the protocol of an animal in each group will serve to point out the salient features.

ABSTRACT OF PROTOCOLS

GROUP 1.—The basilar artery was tied just above the anterior border of the foramen magnum, and just posterior to the pituitary fossa. The animal, lying on its back, immediately after completion of the ties, exhibited marked extensor rigidity in the forelegs; the pinna, sneeze and conjunctival reflexes were brisk, and the withdrawal reflexes were present. The pupils reacted to light with a pseudo-affective cry. When a noise was made the animal's ears were erected. Placed on the floor in the ventral position it acted similar to a labyrinthless animal. There was marked flexion and clasping of the forelegs. When the head was passively extended and dropped it fell to the floor. The animal was turned on its side and the head passively rotated with the occiput down, and marked rigidity in extension occurred in all extremities; when the head was passively flexed the forelegs were suddenly and strongly flexed, so forcibly as to grasp the face with unsheathed claws. Occasionally it struck with the forelegs, as in clawing. There was no spontaneous effort at progression, but while it lay on its side running movements often occurred, and were so pronounced that it was difficult to get the animal posed in any position. When placed on its back it attempted to roll forward into a ventral position. When suspended there was marked rigidity in the hind legs in extension, and in the forelegs in partial extension. When the head was passively hyperextended the forelegs were likewise strongly extended. When the animal was rolled from one side to the other it thrust both forelegs to the side toward which the head was turned, and more marked extension appeared in the opposite hindleg. At times the head was spontaneously lifted from the floor and hyperextended, with ensuing marked rigidity in extension of the forelegs. Then the head rolled to the side and flexed with ensuing strong flexion of the forelegs. This animal lived for thirty-six hours, retaining all the foregoing characteristics. To recapitulate, we had an animal in which there was no predominance of extensor tone of the neck, although the tonic labyrinthine reflex was present. The position of the forelegs was in strong flexion (kangaroo position) unless the head was passively extended. The animal was very active, many reflexes could be elicited, forward rolling was present, and the animal attempted to right itself when rolled from side to side. Although lasting patterns of rigidity in extension, as in an ordinary decerebrate animal, could be produced, lasting patterns in flexion occurred.

The area destroyed in this preparation cannot be accurately described because of the impossibility of injecting the segment of the basilar artery between the ligatures. Certainly, as is shown by Stopford,

complete anemia of the segment does not occur as some vessels from the superior cerebellar artery enter from above, and from the inferior cerebellar and probably basilar from below. The ventral portion of the pons is anemic, as is the inferior portion of the cerebellum. A decerebrate preparation is present in the sense of a production of decerebrate rigidity in extension, but some impulses from above the level of the uppermost ligature and from the superior part of the cerebellum may pass through. Such impulses as do pass through are sufficient to inhibit the predominance of extensor tone of the labyrinth when the animal is allowed to remain in the position it spontaneously assumes. When the head is passively hyperextended, however, the combined neck and labyrinthine tonic reflexes predominate, and rigidity in extension occurs in the forelegs.

GROUP 2.—Another series of animals was decerebrated by a ligation of the basilar artery as high as possible, and ligation of both carotids. Lying on its back, following the operation, the animal gradually extended the legs, more markedly the forelegs. When taken from the operating board and placed on its side, tone became greater in the uppermost side; the head then anteflexed and strong flexion of the forelegs and paws occurred with unsheathed claws, so that at times the animal grasped its own face and was unable to release it, at which time it cried out. When turned from side to side rapidly it attempted to right itself by rapid movements of the forelegs, that is, if turned from left to right the left foreleg abducted and extended. When the animal was turned on its back with the head spontaneously flexed, and was then released, it attempted to roll forward as if to get into a ventral position. When placed on its abdomen, the forelegs were in a position similar to the kangaroo position of flexion. Frequently it attempted progressive movements. When placed in a cage it crawled along the wall, then sprang forward, clasping the net of the cage, and crawled up the net until the head became extended, when rigidity in extension in the forelegs occurred, and the animal remained immobile until gravity overcame its grasp on the cage wall, and it toppled backward to the ground. Then the animal righted itself and again crawled and sprang. Springing can be evoked by placing the animal in the position of sitting on its haunches, and by holding it up by the forelegs and lowering it to its hindlegs. When it was placed on its abdomen, and pulled backward it attempted to crawl forward. When placed on its side and the head passively rotated with occiput down a marked rigidity in extension occurred in the forelegs, demonstrating an intact labyrinthine tonic reflex. When the head was turned chin to the floor marked running movements and clawing occurred, and the animal sprang forward, righting itself, and crawled until it was directly on its abdomen when it rested, with tone apparently normally distributed. Tonic neck reflexes could be elicited. When the animal was suspended marked running movements occurred, so wild at times that it caught its hindlegs by the claws of the forepaws. When now the head was passively extended the forelegs extended and a typical decerebrate rigidity in a caricature of standing was produced. Withdrawal, crossed reflexes, clawing, sneezing, pinna and vibrissae reflexes were present.

This animal was anatomically decerebrated, showing the recognized attitude of decerebrate rigidity in extension in the forelegs, which had at times normally distributed tone, decerebrate rigidity in flexion in the

forelegs, and spontaneous crawling, springing and righting movements. Anatomically the animal differed from the ordinary anemic decerebrate preparation only in a higher level of decerebration. The superior part of the cerebellum was functionless. It is apparent that decerebrate rigidity in extension or flexion and righting movements may occur whether either superior or inferior part of the cerebellum is functionless.

Several decerebello-cerebrate animals were prepared by ligation of the basilar and posterior inferior cerebellars and carotid arteries. Unfortunately, these were unverified because of failure of staining after intravitam injection. However, two observations may be mentioned now and verified later. The labyrinthine tonic reflexes were very pronounced. When the head was turned with the occiput down, the forelegs were thrust forward suddenly and with great force, unsheathing the claws. The head frequently was hyperextended to a degree not observed in other animals. The rigidity in extension of the forelegs was pronounced when that pattern was released. Otherwise, the animal acted as a high tie animal. It would seem that the cerebellum inhibits labyrinthine tonic reflexes.

COMMENT

Occasionally, flexor rigidity has been noted in decerebrate animals by other workers. Bazett and Penfield,[3] working on chronic as well as acute decerebrated preparations of the cat, found that an extensor rigidity might alternate with one of flexion, and that the more chronic the preparation the more tendency there was for a flexed position to appear. "In the chronic preparations they (i. e., rigid flexed positions) are a comparatively common occurrence. . . ." They stated that "the term decerebrate rigidity is generally taken to mean extensor rigidity. But in completely decerebrated specimens another type of rigidity sometimes appears, tonic contraction of the flexor aspect of the limbs. . . . It would be more accurate, therefore, to speak of extensor and flexor rigidity as two types of decerebrate rigidity." Our experiments on labyrinthless decerebrate animals and double and high tie animals corroborate this. Not only do these two types exist, but they can be produced at will in the same animal on proper stimulation. The flexor position of the forelegs was constantly observed by Magnus when in an otherwise normal animal the labyrinths were destroyed and the posterior roots of the cervical nerves were cut. He called this the kangaroo position. When he now decerebrated the animals the forelegs developed a rigidity in extension. It would appear that the reflex activities above the level of the tonic reflexes tend to impose on the animal a position of flexion.

3. Bazett, H. C., and Penfield, W. G.: A Study of the Sherrington Decerebrate Animal in the Chronic as Well as the Acute Condition, Brain **45**:185 (Oct.) 1922.

Attention must be called to a certain fixation of position. When in a labyrinthless or high or double tie animal a rigidity in extension was produced and continued by proper position of the head, flexor patterns became more difficult to produce in direct ratio to the length of time extensor rigidity had existed. This may explain, in part, the failure of the other observers to note the flexor attitude, as attention was chiefly directed to the production of the well recognized position of extensor rigidity in the forelegs.

In the preparation of the animal decerebrated by a high tie, and in that in which a segment of the basilar artery was excluded from the circulation, lasting patterns of rigidity in flexion in the forelegs occurred with the labyrinths intact; in the former the superior part of the cerebellum, and in the latter the inferior portion, was functionless. Removal of the whole cerebellum in an ordinary anemic decerebrate animal did not affect the pattern nor degree of rigidity. Unverified examples of removal of the cerebellum in high tie animals seemed only to augment the labyrinthine tonic reflexes. Common to both preparations here described was the inclusion of higher levels of the pons in the functioning part of the brain stem and in the high tie animal includes a small part only of the red nucleus in which is placed the center for the labyrinthine righting reflexes and body righting reflexes acting on the head. The neck righting reflexes extend as far down as the pons and the upper part of the bulb. The others lie at the level of the red nucleus. The absence of destruction of tissue beyond the borders of the anemic decerebration probably accounts for the constant production of animals possessing some part of the righting reflexes. It is obvious that the labyrinthine, neck, and body righting reflexes were not totally intact. Parts of each, however, must have function. Although the animals were unable while lying on the abdomen to hold the head in a normal position, they attempted to assume a ventral position when placed on their backs. Although the springing reflex was not produced as in the thalamic animals by dorsal flexion of the head, its spontaneous occurrence, we feel, distinguishes it from a bilateral extensor thrust of spinal origin. The attempts to right the position of the body when rolled from side to side, the spontaneous crawling, rarely walking, and crawling forward when pulled backward, all seem to us to point to the partial conservation of fragments of righting and other reflexes than tonic.

When righting and tonic or standing reflexes are intact a normal distribution of tone is present. When tonic reflexes are destroyed in an otherwise normal animal flexor patterns (kangaroo position) predominate. When the righting reflexes are only partially destroyed in a decerebrate animal both flexor and extensor rigidity may be produced

by suitable stimulation (high tie and double tie animals). When the righting reflexes are absent in a decerebrate animal labyrinthine tonic reflexes predominate, and extensor rigidity occurs (ordinary decerebrate animals). When in such an animal the labyrinths are destroyed flexor rigidity usually spontaneously occurs, but extensor rigidity can then be produced by the neck tonic reflexes.

DISCUSSION

ON PAPERS OF DRS. KRAUS, BROCK AND WECHSLER, AND POLLOCK AND DAVIS

DR. G. SCHALTENBRAND, Hamburg, Germany: I am especially interested in Dr. Kraus' description of the development of the erect posture in man. I did not know about his work when I made an investigation of the development of the different righting reflexes in man in Professor Nonne's clinic in Hamburg.

I examined more than 120 children of different ages according to the schema worked out by Magnus and de Kleijn. The chief differences between all of these righting reflexes of man and animals are due to the vertical posture of the human body in contrast to the horizontal posture in animals. In the ontogenetic development of the posture and of the righting reflexes, man passes through stages comparable to those in animals. The most striking changes in man occur during the first years of life and are alterations of those movements which are necessary to stand up from the recumbent position. In these movements all the righting reflexes work together. An animal laid on its back will return to the normal position by rotating the whole body around its axis. It begins with the rotation of the head, followed by the rotation of the shoulder girdle and finally of the pelvis. I found this same movement in infants of 1 to 2 years. During the second year, children learn to reach the upright position, but they do this after having made the rotation of the body described above. Adults stand up in a different way. They elevate the head and trunk from the floor until they sit, without first rotating the body, and then arise from this position. The torsion movement of the "primitive type" is replaced by the symmetrical movement of the adult type during the first five or six years of life. The primitive form of standing up returns in adult persons, however, who have paresis or paralysis of muscles, especially of the abdominal wall; for instance, in progressive muscular dystrophy. It is of interest that the primitive form of standing up may also occur in adult persons with intact musculature as a symptom of brain tumor. I observed this in three verified cases of glioma of the temporal and parietal lobe which I saw in the clinics of Professor Brouwer in Amsterdam and Dr. Cushing of Boston. I saw the same movement occurring in an unconscious epileptic patient between the fits, and in the case of an idiotic child and finally in some children who had cerebellar tumor and a large hydrocephalus.

As to the interpretation of the parkinsonian syndrome, my conclusions are different from those of Dr. Kraus, Dr. Wechsler and Dr. Brock. This is probably due chiefly to differences of terminology. These investigators speak of the "righting reflex," and I think that they use the term righting reflex in too broad a way, making it embrace much more than Magnus and de Kleijn do. I found that the righting reflexes, especially the movements of standing up, were performed according to the adult type in a series of cases of chorea

and a series of cases of parkinsonism. Only a few patients with severe parkinsonian disease were unable to stand up from the floor. Most patients with parkinsonian disease are very slow, so that I had to wait for a long time until they finally found the impulse to stand up. It seems to me that it is not a lack of certain movements and of certain reflexes, but rather a lack of spontaneity with slowness of all movements, that are the outstanding features of these patients.

If the essential feature of the parkinsonian state is more or less a reversion to the quadripedal status of the lower mammal, as Dr. Kraus thinks, we should find parkinsonian states as a pathologic condition in mammals because they are already in this position. The picture of parkinsonism as a pathologic condition can be present in mammals. It has been shown by Lewy in lower animals and by Mella in monkeys that manganese intoxication produces paralysis agitans with flexion, tremors and slowness. I have studied the effects of bulbocapnine, which is known to produce catalepsy, and I saw in apes, dogs and cats a state consisting of flexion, slight rigidity, occasional tremor and a striking lack of movements. I think that such a picture may be regarded as a parkinsonian state. Therefore, I came to the conclusion that diseases of the chorea and parkinsonian type are conditions, not of the righting reflexes themselves, but of some higher motor regulations that are superior to the righting reflexes. I believe that these higher regulations are present in man as well as in higher mammals.

Dr. B. Brouwer, Amsterdam, Holland: The work of Magnus and de Kleijn has influenced also the neurologic clinic in Amsterdam, perhaps much earlier than your clinics, because we are so near that we naturally learned about it quicker. I have seen, for example, these reflexes in a case of encephalitis of the forebrain (with postmortem examination).

I would emphasize one point. I have heard that there is a tendency to localize the center of erectness of man in a part of the brain immediately before the midbrain or in the substantia nigra. You have been told about paralysis agitans, and now I should like to say that when there is a lesion in a special part of the brain, causing a disturbance of function, it is not permissible to localize this function in the part of the brain in which the lesion is found. For example, when there is hemianopia, caused by destruction of the calcarine cortex, it cannot be concluded that the function of sight is localized in this part of the brain. I agree with you that the part of the central nervous system localized before the midbrain is one of the centers, but the chief centers of erectness must lie in the cortex of the brain. This function is such a highly organized one and appears so late during phylogenetic development, that it is evident that a great deal of the cortex has an influence on this function.

Dr. Sidney I. Schwab, St. Louis: I should like to ask Dr. Kraus whether it is possible for posture to disappear or tend to disappear in the reverse direction from the phylogenetic. In St. Louis, a case of encephalitis is being studied in which the patient is losing his erect posture gradually but in an extended position. There is also a case of dementia praecox in which the defective posture is in the hyperextension rather than in the flexion position.

Dr. W. Penfield, New York: The method that Dr. Pollock and Dr. Davis have provided seems an important one. There has been some criticism of it because the exact level of the decerebration is difficult to determine. Decerebrate animals should be judged physiologically. If they present, as the animals of these speakers did, decerebrate attitudes, they are decerebrate animals and can be compared directly with those prepared by the standard

method or what Dr. Pollock calls the mutilating operation. All of the animals should be controlled afterward by careful and painstaking microscopic examination, however, to determine just how high the level of paralysis really is.

From the point of view of the red nucleus, it has been evident, of course, from the work of Magnus and in some of the animals that I had an opportunity to study with Dr. Bazett, that the red nucleus can be entirely absent in preparations that show a decerebrate posture; so that the view of those who felt that the red nucleus was the source of the tone in decerebrate rigidity is not borne out in animals.

Transverse section below the hypothalamus liberates some mechanism of posture maintenance which Magnus has analyzed and divided into component parts. Amputation through the upper part of the pons removes that mechanism, but it cannot be said, therefore, that there are definite centers of posture between the pons and hypothalamus. One may be only interrupting long arcs or circuits which pass through the midbrain at that point. Some of those arcs probably pass through the red nucleus; others are lower.

To pass from animals to man, from an anatomic point of view, is difficult, because, as is known, function in man has migrated forward in a general way. Although it is impossible to compare animals with man anatomically, from a physiologic point of view it is possible to assume, for instance, that hemiplegic rigidity is similar to decerebrate rigidity. The hemiplegic position is not exactly the pattern which has been described by Wilson as the decerebrate attitude in man, but in man the erect posture is not normally accompanied by extension of the forearms. It is perhaps more likely to accompany flexion of the forearms or no pattern of postural tone.

In the frog the decerebrate attitude is not one of extension but one of flexion of the hind legs, and that is his normally erect attitude. So that, *a priori,* the hemiplegic flexion of the forearm in man could well be considered the decerebrate attitude, supposing that postural localization has migrated forward in the human brain.

Dr. A. T. Mussen, Baltimore: Since Magnus and de Kleijn's work on the Körperstellung, the conclusions of which were based largely on the experiments of Rademaker on decerebrate animals, the red nucleus appears to have been generally accepted as the nucleus responsible for the righting reflexes. In the last three papers references have been made to this work, and the writings of Walshe and Wilson support it.

The results of my experiments on the red nucleus, which were reported at the meeting of the American Neurological Association last year, do not confirm this view. In one experiment the posterior region of a single nucleus was destroyed. A crossed degeneration of the rubrospinal tract resulted. There were no symptoms. Another specimen in which the lesion involved the posterior regions of both nuclei, with a complete bilateral degeneration of the tracts, also showed no symptoms. When the lesion affected the anterior region of the nucleus parvocellularis, from which a well marked tract of degenerated fibers—I believe not previously described—could be followed to the opposite cervical region, there was complete loss of the "righting reflexes"; after some days, these reflexes again appeared.

The remarks of Dr. Brouwer support the opinion expressed in my original paper that the righting reflex is not controlled by any particular nucleus, but that it is probably regulated by the coordinated action of complete cerebration. It is necessary to remember that in decerebrate animals one is dealing

with specimens in which only the brain stem remains, and that it is difficult to determine the function of a nucleus when it is removed from the influence of the higher centers.

Dr. S. Philip Goodhart, New York: The importance that Drs. Kraus, Wechsler and Brock have given to the term "postural mechanism" and their description of that mechanism as one possessed of special and independent function is of much clinical significance.

That posture and tone are two separate manifestations, each with its own controlling mechanism but, generally speaking, each independent of the other, I have long been convinced. A study of several of my cases in the post-encephalitic groups has emphasized my previous observations. Since talking with the authors of the papers just presented and reading their work, I have restudied more carefully some of the subjects that I have as film reproductions, and my previous impressions have been confirmed. For example, in a young man and a young woman, both of whom you have seen, with postencephalitic parkinsonism and a definite though only moderate increase in tone, the postural design is every now and then suddenly broken up, the patients "fold up" like a jack-knife, and "fall together," at least so it appears to the eye; tone apparently remains unchanged during these periods of postural disintegration.

The first two cases referred to perhaps answer the pertinent questions of Drs. Wechsler and Brock as to whether the righting mechanism is so dependent on tone as to make it impossible for the patient to stand upright because of abnormal tone. In my patients though the tone was only moderately increased, there was absolute loss of the righting mechanism. The postural design was gradually disrupted. It appeared as though the coordinated, the synthetized elements, which determine the upright position had become disintegrated. The patients could not rise unassisted.

Dr. Walter M. Kraus: The short time at my disposal permits me to make only one comment, which is that though man and monkeys have descended from a common and remote ancestor, it is impossible to identify either normal or postural manifestations in monkeys with those of man. I think it would be difficult to correlate Dr. Mella's and Dr. Schaltenbrand's work on monkeys with observations on man.

I am in entire accord with what Professor Brouwer has said. It is apparent that for a center to be active, it must be activated. It is certain from our knowledge of our human infancy that when walking began, the cortex must have activated the center of erectness. This does not in the least vitiate the fact that the center must be infracortical. Just as spinal centers are activated from the region of the midbrain in decerebration, so centers having to do with erectness are unquestionably activated by the cortex.

Dr. Schwab's question interests me, and the problem has puzzled me for a number of years. I expect to take up the study of this matter this winter. At present, all I can point out is that the dorsal and ventral axial muscles are definitely controlled in the righting reflex underlying erectness. When an excess of erectness occurs, and when we wish to increase our usual erectness — as in assuming the military position — we increase voluntarily the action of our dorsal muscles. Why under certain pathologic circumstances this posture occurs, I cannot explain; I have seen it frequently. All that can be said is that it represents the reverse of a dissolution of erectness.

Dr. Penfield's remarks also interested me because I have been a student of his paper with Dr. Bazett and have read it many times. I think the contribu-

tion is so large that it would be impossible to touch on more than one point in passing. That is the point that Dr. Pollock has emphasized, that flexion and extension are both activated in decerebrate animals.

Another point, the neurobiotactic pulling up of posture centers toward the fore part of the brain does not, I think, indicate that the posture of hemiplegia or parkinsonism is identical with that of the decerebrate arm. This new posture is entirely different.

DR. LEWIS J. POLLOCK: The sum of our knowledge of a decerebrate animal is the result of the work that has followed Sherrington's classic preparation. I do not mean that Sherrington's is the only preparation that can be made, and it does not mean that any subsequent preparations are faulty.

It is obvious that this preparation has at least permitted us to determine regularly the existence of not only the decerebrate rigidity and extension but also that of flexion and other reflexes in the brain stem. The level of the decerebration can be accurately determined, of course, by intravital injection of methylene blue through the general circulation; this is done in all of the animals used in this laboratory, and there is always a clean-cut line between that part which is stained and that which is not.

Unfortunately, owing to the length of time required in the experiments that we have done to date, we have not dealt with chromic preparations, but we shall at some time in the future.

Certainly all of the red nucleus can be destroyed and decerebrate rigidity produced. That is easily understood in relation to our interpretation when we say that when the righting reflexes are absent, the tonic standing reflexes produce a position of extension in the forelegs.

Dr. Mussen's discussion is important because it emphasizes the necessity for distinguishing between the reactions of a normal brain or one that has a slight lesion and the reaction of a brain with only a slight part of the brain stem remaining. Only a few activities reside in that brain, and it is impossible to judge from the remaining activity what exists above.

I wish to state in conclusion only that a decerebrate animal is not synonymous with decerebrate rigidity. A decerebrate animal has many reflex activities, some of which have been described.

FRIEDREICH'S ATAXIA

CLINICAL AND POSTMORTEM STUDY OF TWO CASES IN BROTHERS AT DIFFERENT STAGES OF THE DISEASE *

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Although reports of the occurrence of Friedreich's ataxia in siblings are not uncommon, rarely has an opportunity been presented for the clinical study during the different stages of the disease and of the spinal cord lesions in such cases. The cases here described were those of two brothers, fullblooded negroes, each of whom was 8 years of age when the symptoms were first observed. The duration of life, from the time of the onset of the illness, was three years in the case of one brother, and seven years in the case of the other.

The case of the elder brother was described by Lloyd and Newcomer,[1] in 1921. At that time the younger brother was still alive. His death, in October, 1924, furnished the opportunity for the present comparison of the two cases.

REPORT OF CASES

CASE 1.—*Family History.*—The parents were living and in good health. There was no history of nervous disease in the relatives or the antecedents, as far as could be ascertained. The mother had had one miscarriage, but her blood Wassermann reaction was negative. There were three other children in the family. The eldest daughter had had convulsions when 2 years of age but had since enjoyed good health. One brother, who died in infancy, had had convulsions. The youngest daughter was in good health.

J. C., the elder brother, was 11 years old at the time of his death. Dr. Lloyd[1] reported: "He was born in normal labor, and walked and talked at the usual age. He had convulsions in infancy and the usual diseases of childhood. He went to school for a while and did well. The ataxia began when the boy was 8. He became awkward in the movements of his legs, failing on his knees when attempting to walk. Scoliosis was present. When he was admitted to the hospital, in the autumn of 1919, his gait was very ataxic; he kept his feet far apart, swayed violently, and required assistance in walking. It was not the gait of locomotor ataxia. He had the Romberg sign, and well marked adiadokokinesis. The finger-to-nose and heel-to-knee tests showed ataxia. The knee and Achilles reflexes were absent, and there was no Babinski reflex. The abdominal and cremasteric reflexes were preserved. Superficial sensation in all its modes was normal; that is, to touch, pain, heat and cold. There was apparent loss of ability to recognize a tuning fork held on the

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1. Lloyd, J. H., and Newcomer, H. S.: A Case of Friedreich's Ataxia, Arch. Neurol. & Psychiat. **6**:157 (Aug.) 1921.

radius, ulna or tibia. There was no shortening of the plantar arch, no hammer-toe, as reported in some cases; no muscular atrophy, no pain, no girdle sense, no loss of sphincter control. Speech was hesitating and staccato rather than drawling. Vision was normal, the pupils equal, the reaction to light sluggish, the action in accommodation and convergence prompt. Dr. Shannon reported a curious lack of pigmentation in the upper nasal quadrant of the iris of the right eye, also in the upper nasal and lower temporal quadrants of the left iris, suggesting iritic atrophy. On distant fixation, the left eye rotated outward, although on close fixation fair convergence was obtained, indicating a condition of exophoria. This measured about 12 degrees with the Maddox rod. Ataxic nystagmus was not elicited. In the right eye the media were clear, the disk discolored and somewhat atrophic, the margins clearly outlined, the vessels moderately contracted, the macula normal; there were no changes in the periphery of the fundus. The left eye was in a similar condition. A few months after his admission to the hospital, this boy died of influenzal pneumonia."

Histologic Report on Postmortem Examination of Spinal Cord by Dr. Newcomer.[1]—The gross appearance of the cord was not remarkable. Sections from various levels, stained with toluidin blue, showed marked pigmentation of the anterior horn cells. The lateral horns showed some simple chromatolysis. In numerous sections from the dorsal region of the cord, no normal Clarke's column cells were found. These cells were either absent or, when present, were greatly diminished in number and markedly sclerosed. There was considerable gliosis of the column. In the cord, there was fibrosis of the vessels, but no infiltration. Sections stained with phosphotungstic acid hematoxylin gave evidence of considerable gliosis of the posterior columns, which substantially involved, in all the levels, the region of the third myelogenetic system, as described by Trepinski. In this region, the fibers were practically all gone. The involvement of the third myelogenetic system in the posterior columns is shown in the photographs of the Weigert specimens (fig. 1, *D*, *E* and *F*). In the rest of the posterior columns, there was a gliosis of less intensity. The gliosis in the region of the greatest scarring consisted of a dense fibrous mat with rather small, inactive nuclei—a mat of whorls and irregularly arranged glia fibers. There were some spider cells, but none of the columnar fiber arrangement so typical of tabes. In the less involved region of the posterior columns, the gliosis was also largely of fibrous character, but spider cells were more numerous. In all the levels of the cord, there was some gliosis of the crossed pyramidal tract. This gliosis was fibrous in character but contained a number of rather large spider cells. Nerve fiber replacement was appreciable in the center of the tract. At the border of the cord in the region of the dorsal cerebellar tract, there was a certain amount of spider cell gliosis, and the glia fiber strands were heavier and more conspicuous than anywhere else along the border of the cord.

In the region of the direct pyramidal tract, there was some gliosis in association with large spider cells. This slight gliosis of the direct pyramidal tract decreased as one progressed toward the dorsal and lumbar regions. There was more gliosis than usual in the region of the anterior commissure. In the gray matter of the cord, there were strikingly more than the usual number of glia cells; many of them were large spider cells, quite active in appearance. The gliosis in the gray matter, both fibrous and cellular, was greatest in the lower regions of the cord. It was less in the posterior horns than elsewhere. There was considerable fiber loss with extensive spindle cell replacement in

the posterior nerve roots and considerable glia fiber invasion of the proximal portions of the nerve roots. The fiber loss was greatest in the cervical region, involving most of the fibers, and decreased gradually to become least in the sacral region, where only a few fibers were gone. The anterior roots were intact. With the acid fuchsin-light green stain, the extensive cord gliosis was distinct, and it was possible to estimate the fiber loss in the direct cerebellar tracts as being about one-sixth. The crossed pyramidal tracts were about the same. The posterior column gliosis was, if anything, less than this except in the region of the third system, in which the fiber loss was practically complete. In the ventral field, more than half the fibers were gone.

Case 2 (from the wards of the Philadelphia General Hospital).—*History.*—M. C., the younger brother, was 9 years old when first admitted to the hospital in October, 1919. His birth had been normal, and he had been a strong, healthy infant, except that he had had two convulsions at the age of 1 year. He walked and talked at the usual age, and had the ordinary diseases of childhood. He started to go to school when he was 7, and did well.

During the latter part of his seventh year, he was stricken with typhoid fever. While he was convalescing, a marked weakness of his legs was noticed. He began to stagger a great deal when walking, and the movements of his arms also became very clumsy. When this condition had progressed for one year, he was admitted to the hospital.

On admission, the patient was fairly well nourished, and there was no apparent weakness or atrophy of the muscles. The speech was without defect, and he could swallow well. However, the gait was unsteady and he would shuffle his feet in order to maintain his balance. This disturbance was not increased when the eyes were closed. Awkwardness of the ataxic type was brought out by the finger-to-finger, finger-to-nose, heel-to-knee and heel-to-toe tests. Both patellar and Achilles reflexes were absent, as were also the left biceps and triceps reflexes. The abdominal and cremasteric reflexes were preserved. There was no Babinski reflex and no ankle clonus. Sphincteric control was good. There was no shortening of the plantar arch and no hammer-toe. The patient had flatfoot with slight eversion. Sensibility for touch, pain and temperature was undisturbed, but vibratory sense was lost over the tibia, radius and ulna.

The patient left the hospital, April 1, 1920, but was readmitted, Sept. 22, 1923. During this interval, his condition had become progressively worse. He had become helpless and bedfast, and now complained of difficulty of articulation and swallowing.

During the next twelve months, the bulbar symptoms became more pronounced. The patient was unable to speak above a whisper; he could not purse his lips, and saliva escaped from his mouth. Mastication and swallowing became increasingly difficult, and respiration was labored and irregular. Partial opthalmoplegia, slow lateral nystagmus, loss of the pupillary light reflex, optic atrophy and pigmentation of the optic disks were observed. The tongue was weak and showed a fibrillary tremor. All the deep reflexes were absent. The corneal and abdominal reflexes remained. There was no Babinski reflex or ankle clonus. Sensation for touch, pain and temperature remained unimpaired. There was considerable wasting of all the muscles except those of the face. Right scoliosis and lumbar lordosis were present. Both feet were everted and arched, but there was no hammer-toe. The patient was capable of a few slow, feeble movements of the extremities, but these were characterized by marked ataxia. The weakness seemed to be attributable chiefly to extreme muscle

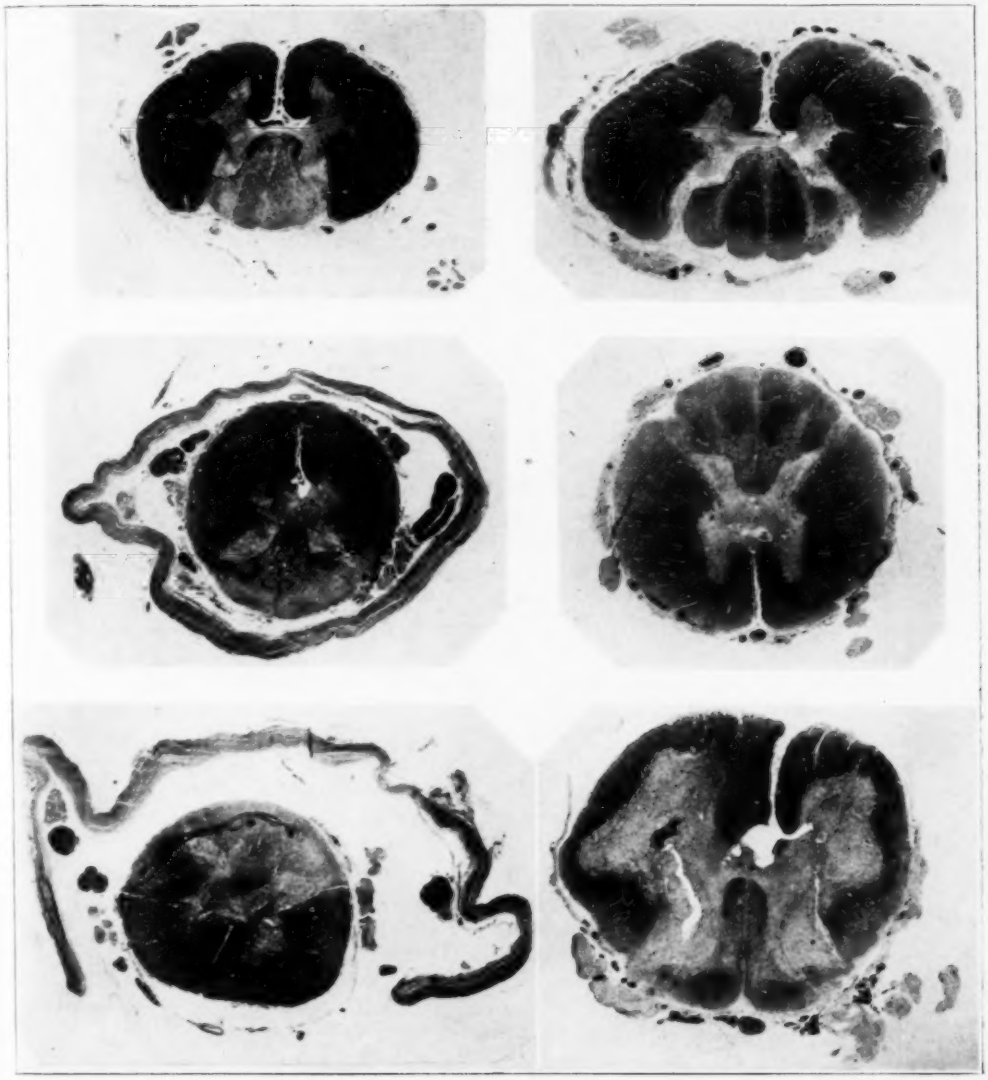

Fig. 1.—Weigert sections of different levels of the cord, for the purpose of comparing the changes in the two brothers. *A*, *B* and *C* are from the younger brother (the author's case). *D*, *E* and *F* are from the elder brother (the case previously reported by Lloyd and Newcomer[1]). *A* and *C*, cervical cord; *B* and *E*, thoracic cord; *C* and *F*, lumbar cord. The more extensive degeneration in the present case should be noted (*A*, *B* and *C*). The case of Lloyd and Newcomer (*D*, *E* and *F*) shows involvement of the third myelogenetic system in the posterior columns. The peripheral paleness is an artefact.

atony. Control of the bladder and bowels was retained. The mental condition remained fairly good. Death occurred in October, 1924, from bronchopneumonia.

Necropsy Report.—Necropsy permission was limited to the spinal cord. This was unusually small and was flattened anteroposteriorly. On gross examination, the dura mater and the pia-arachnoid appeared normal.

Sections were taken from various levels of the cord and stained with the following agents: hematoxylin-eosin, phosphotungstic acid hematoxylin, Weigert's myelin sheath stain, Bielschowsky's stain, the Alzheimer-Mann stain, the Marchi stain and toluidin blue.

Microscopic Study of Spinal Cord.—Cervical region: With the Weigert stain, the posterior columns showed a lightened area corresponding to the tracts of Goll and Burdach. At the anterior apexes of Burdach's tracts, adjacent to the posterior commissure, a group of fairly normal fibers remained (fig. 1, *A*). The posterior roots were sclerosed; Lissauer's tracts were much affected.

The lateral and anterior columns were of normal appearance, and the anterior roots were intact.

The glia was greatly increased in the middle zone of the posterior column, and there were a number of clumps of glial fibers arranged in a whorl-like manner about the blood vessels (fig. 2). Increase in glia was noted also in the direct cerebellar and in the crossed pyramidal tracts.

Longitudinal sections through the middle of the posterior column, stained by the Bielschowsky method, showed marked destruction of the fibers (fig. 3). Here the degeneration was most intense in the central part, corresponding to Goll's tract; on either side of this area, the fibers appeared more normal. Many of the affected fibers were swollen and had nodular enlargements. Some were devoid of myelin covering, and others appeared to be fragmented.

The anterior horn cells were diminished in number. Most of those remaining were in various stages of degeneration (fig. 4). Some were pale and swollen, with scarcely visible nuclei; others were small, shrunken and deeply stained, with eccentric nuclei. A few had been reduced to mere shadows. Large deposits of lipoid material appeared in many of the cell bodies.

Dorsal Region: Sections from the upper dorsal cord presented almost the same changes as in the cervical region. In the lower dorsal region, however, the sclerosis of the posterior columns was more uniform and widespread (fig. 1, *B*). The group of intact fibers dorsal to the posterior commissure remained as described above for the cervical region (fig. 1, *A*), but here it was larger and triangular in shape. In the lateral and anterior columns, evidence of slight sclerosis was present, the lightened area corresponding to the crossed pyramidal and cerebellar tracts.

The glial increase was most marked in the middle part of the posterior column; in the remainder of the circumference of the cord, it extended inward from the periphery. Clarke's cells were diminished in number, only one or two appearing in each section. They were swollen and lightly stained, with pale eccentric nuclei.

Lumbar Region: The posterior columns showed less degeneration in this region than at the higher levels (fig. 1, *C*). Here the lightened area in the Weigert sections was more marked in the dorsolateral portions. Sclerosis of the lateral and anterior columns was apparently slight.

The area of glial proliferation in the posterior column extended dorsad from the posterior commissure so as to form one central and two lateral projections, resembling somewhat the letter W. Masses of wavy hairlike glial

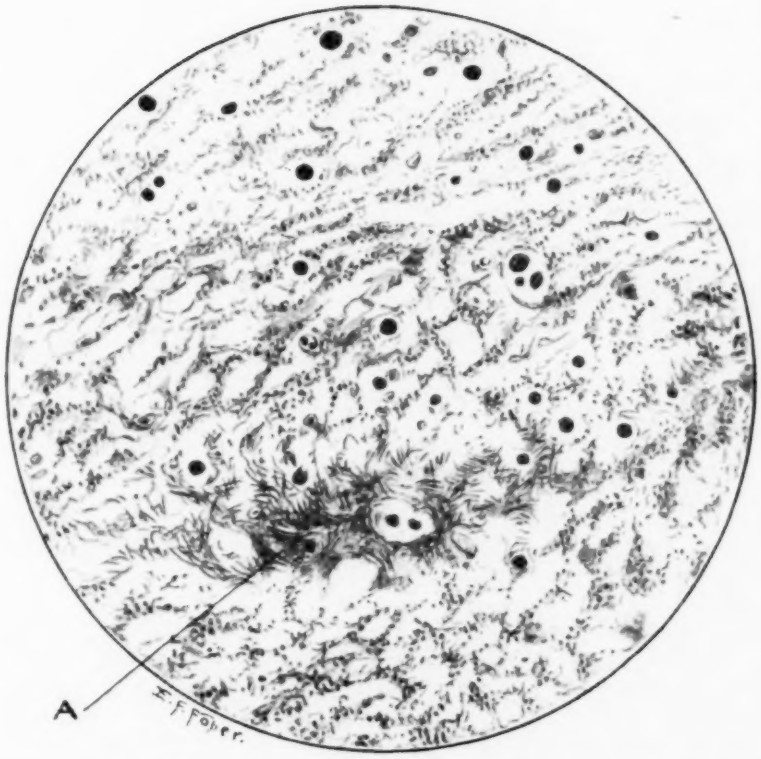

Fig. 2.—Camera lucida painting from oil immersion view showing whorl of neuroglia proliferation surrounding small blood vessels.

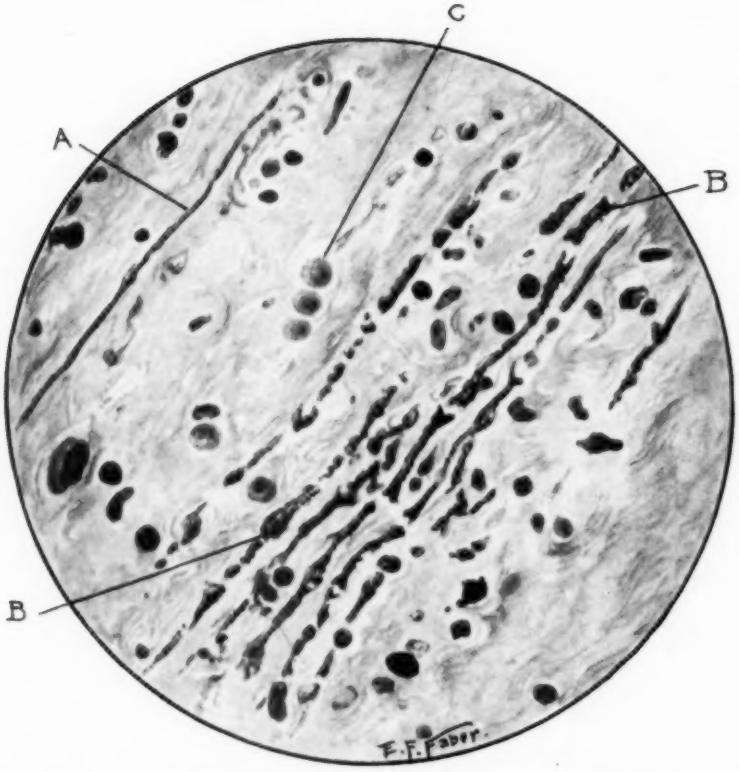

Fig. 3.—Camera lucida painting from longitudinal section through posterior column, showing swelling and fragmentation of fibers.

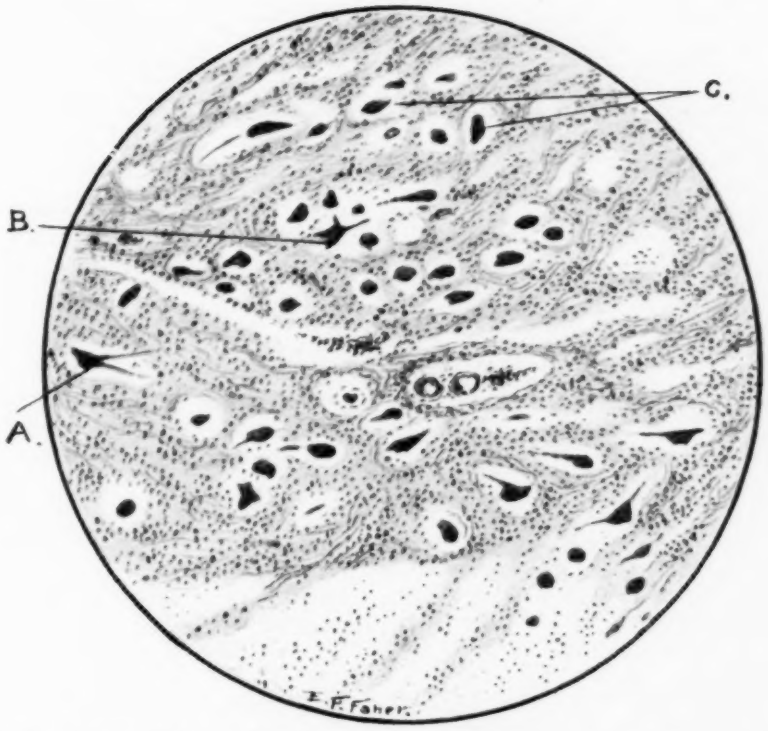

Fig. 4.—Camera lucida painting from anterior horns of lumbar region, showing degeneration of cells (low power magnification).

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fibers were visible in the darkened area. The glia increase in the lateral and anterior columns was not marked.

The anterior horn cells in the lumbar enlargement were less than normal in number and presented marked degenerative changes.

Summary of Spinal Cord Lesions.—(1) The cord was small; (2) there were sclerosis of the posterior columns, more marked at the higher levels; slight sclerosis of the lateral columns, diminishing toward the medulla oblongata; slight involvement of the anterior columns; (3) the glial proliferation was most marked in the posterior columns showing whorl-like masses of glial fibers; (4) numerical diminution and degeneration of the anterior horn cells and the cells of Clarke's column were found.

TABLE 1.—*Age at Onset and Duration (Thirty-Eight Cases)*

Author	Age at Onset	Duration in Years
Auscher (Compt. rend. Soc. de biol. **2**: 475, 1890)	25	4
Barker (Trans. A. Am. Phys. **18**: 637, 1903)		
Case 1	18	12
Case 2	18	12
Dejerine and André-Thomas (Rev. neurol. **15**: 41 [Jan. 30] 1907)	12	23
Fergus (Glasgow M. J. **6**: 193 [Oct.] 1917)	14	..
Hess (M. Clin. N. Amer. **5**: 1749 [May] 1922)	8	..
Hoffmann (Deutsche Ztschr. f. Nervenh. **60**: 179 [Aug. 17] 1918)		
Case 1	25	4
Case 2	19	19
Hunt (Am. J. Obst. **62**: 125 [July] 1910)		
Case 1	23	..
Case 2	8	..
Case 3	5	..
Case 4	3	..
Lambrior (Rev. neurol. **25**: 57 [Jan. 30] 1913)	12	4
Letulle and Vaquez (Comp. rend. Soc. de biol. **2**: 21, 1890)	10	11
Litchfield, Latham and Campbell (M. J. Australia **1**: 135 ([Feb. 1] 1917)		
Case 1	6	2
Case 2	6	8
Lloyd and Newcomer (Arch. Neurol. & Psychiat. **6**: 157 [Aug.] 1921)	8	3
Menzel (Arch. f. Psychiat. **22**: 160, 1891)	28	18
Meyer (Brain **20**: 276, 1897)	45	22
Mott (Path. Lab. London County Asylum **3**: 180, 1907)	16	21
Müller, W. (In. Diss., Würzburg, 1907)	1½	16
Case 1	8	11
Case 2	17	8
Müller, W. (In. Diss., Würzburg, 1907)	1½	16
Osnato (New York M. J. **107**: 204 [Feb. 2] 1918)		
Case 1	14	..
Case 2	9	..
Case 3	12	..
Case 4	12	..
Case 6	8	..
Case 7	10	..
Pfeiffer (Arch. Neurol. & Psychiat. **7**: 341 [March] 1922)	14	15
Philippe and Oberthür (Rev. neurol. **9**: 971, 1901)		
Case 1	24	10
Case 2	2½	15
Pic and Bonnamour (Nouv. iconog. de la Salpêtrière **17**: 126, 1904)	9	30
Purser (Dublin J. M. Sc. **143**: 81 [Feb.] 1917)	16	..
Rennie (Brit. M. J. **2**: 129 [July 15] 1899)	11	1½
Simon (Progrès med. **6**: 145 [Sept. 4] 1897)	2½	15
Spiller (J. Nerv. & Ment. Dis **37**: 411 [July] 1910)	15	37
Thomas (Bull. Johns Hopkins Hosp. **24**: 121 [April] 1913)	10	..

Comment.—In regard to the clinical course of the disease in the two brothers, the points of similarity are of considerable interest. The age at onset was the same in both cases; that is, 8 years. Both patients presented marked ataxia of all extremities, loss of the deep reflexes, scoliosis and pigmentation of the retinal disks. There was no Babinski

reflex in either case, and in both the abdominal reflexes were preserved. The younger brother, however, survived four years longer, and presented the additional symptoms of nystagmus, ophthalmoplegia, complete optic atrophy, loss of speech, difficulty of swallowing and atrophy and extreme hypotonus of the musculature.

The extent of the lesions found in the two cords corresponded, in some respects, to the stage of the disease. From figure 1, a comparison of the cord lesions in the two cases can be made. In the case of shorter duration, the cord was of normal size, and the sclerosis was more or

TABLE 2.—*Eye Symptoms (Thirty-Eight Cases)*

Author	Eye Symptoms
André-Thomas and Durupt [*]	Nystagmus
Auscher [9]	Nystagmus; Argyll Robertson pupils
Barker, case 1	Optic atrophy; ophthalmoplegia
Barker, case 2	Ptosis
Blocq and Marinesco †	Slight nystagmus
Dejerine and André-Thomas	Nystagmus
Hess	Late nystagmus
Hoffmann, case 1	Nystagmus
Hoffmann, case 2 [10]	Nystagmus; congenital bilateral ptosis
Hunt, case 1	No disturbance
Hunt, case 2	Slight nystagmus
Hunt, case 3	No disturbance
Lambrior [11]	Nystagmus
Letulle and Vaquez	Nystagmus
Lhermitte and Artom ‡	Nystagmus; Argyll Robertson pupils
Litchfield, Latham and Campbell, 2 cases	Late nystagmus
Mackay [6]	Nystagmus
Menzel	Sluggish reaction to light and in accommodation
Müller, E.	None
Müller, W.[2]	Nystagmus
Osnato, case 1	Internal strabismus
Osnato, case 2	No definite disturbance
Osnato, case 3	No definite disturbance
Osnato, case 4	No disturbance
Osnato, case 6	Ptosis, nystagmus, optic atrophy
Osnato, case 7	Nystagmus
Pfeiffer	Nystagmus
Philippe and Oberthür, case 1	Argyll Robertson pupils; optic neuritis
Philippe and Oberthür, case 2	Nystagmus; blepharoconjunctivitis
Pic and Bonnamour	Anesthesia of left eye; dim vision
Purser	Nystagmus
Rennie [5]	Slight nystagmus
Rütimeyer, case 1 §	Nystagmus
Rütimeyer, case 2	Nystagmus
Simon [8]	Nystagmus; blepharoconjunctivitis and keratitis
Spiller [4]	Nystagmus; pupils unequal
Thomas	Nystagmus

[*] Rev. neurol. **24**: 317 (Sept. 30) 1912.
† Arch. de neurol. **19**: 331 (May) 1890.
‡ Bull. et mém. Soc. anat. de Paris **62**: 556 (July) 1907.
§ Virchows Arch. f. path. Anat. **91**: 106 (Jan.) 1883.

less limited to a certain area of the posterior columns; but, in the case of longer standing, the cord was much smaller, and the sclerosis involved practically all of the posterior columns.

For the purpose of accounting for some of the differences noted in the two cases and in order to compare the cases in a general way with some of those previously reported, a part of the exceedingly voluminous literature pertaining to Friedreich's disease was studied, tabulated and is presented in tables 1 to 7.

ANALYSIS OF TABLES

Age at Onset and Duration.—In the thirty-eight cases recorded in table 1, the youngest patient was between 1 and 2 years old at the onset of the disease;[2] the oldest, 45.[3] The great majority were under 20 when the symptoms first appeared. In only one of the thirty-eight cases did the onset occur after the age of 30.

TABLE 3.—*Reflexes (Forty-Four Cases)*

Author	Upper Limb	Knee and Ankle	Babinski	Superficial
André-Thomas and Durupt	Absent	Absent	Present	.....
Auscher [9]	.....	Absent	.....	.....
Barker, case 1	Active	Active	.....	.....
Barker, case 2	Active	Active	.....	.....
Barrett	.....	Absent	Absent	.....
Blocq and Marinesco	.....	Absent	.....	.....
Dejerine and André-Thomas	.....	Absent	Present	.....
Fergus, case 1	Absent	Absent	Present	Present
Fergus, case 2	.....	Absent	Present	Present
Hess	Normal	Absent	Present	.....
Hoffmann, case 1	Weak	Active; ankle clonus	Present	.....
Hoffmann, case 2 [10]	Present	Active; ankle clonus	Present	.....
Hunt, case 1	.....	Absent	Absent	Present
Hunt, case 2	.....	Absent	Absent	Present
Hunt, case 3	.....	Absent	Absent	Present
Lambrior [11]	.....	Absent	Absent	Present
Letulle and Vaquez	Absent	Absent	.....	.....
Lhermitte and Artom	Weak	Absent	Uncertain	Absent
Litchfield, Latham and Campbell, case 1	.....	Absent	Absent	.....
Litchfield, Latham and Campbell, case 2	.....	Absent	.....	.....
Lloyd and Newcomer [1]	.....	Absent	Absent	Active
Mackay [6]	Absent	Absent	.....	.....
Menzel	.....	Normal	.....	.....
Meyer	Active	Active	.....	.....
Müller, E., case 1	.....	Absent	.....	Present
Müller, E., case 2	.....	Knee jerks active, ankle jerks absent	Present	Active
Müller, W.[2]	.....	Absent	Absent	.....
Osnato, case 1	Weak	Weak	Present	Present
Osnato, case 2	Weak	Knee jerks absent; ankle jerks weak	Present	Present
Osnato, case 3	Weak	Absent	Present	Present
Osnato, case 4	Absent	Absent	Absent	Present
Osnato, case 6	Weak	Weak	Absent	Weak
Osnato, case 7	Active	Active	Present	Active
Pfeiffer	.....	Absent	Absent	Active
Philippe and Oberthür, case 1	.....	Absent	Absent	Present
Philippe and Oberthür, case 2	.....	Absent	.....	.....
Pie and Bonnamour	Weak	Absent	Absent	Absent
Purser	.....	Absent	Uncertain	.....
Rennie [5]	.....	Absent	Absent	.....
Rütimeyer, case 1	.....	Absent	.....	.....
Rütimeyer, case 2	.....	Active	.....	Present
Simon [8]	.....	Absent	.....	.....
Spiller [4]	Absent	Absent	Absent	.....
Thomas	Absent	Absent	Present	Present

Figures as to the duration of Friedreich's ataxia are of little value, since so few of the patients die of the disease itself. The malady weakens them to such an extent that they readily succumb to intercurrent

2. Müller, W.: Zur pathologischen Anatomie der Friedreichschen Ataxie, In. Diss., Würzburg, 1907.

3. Meyer, A.: The Morbid Anatomy of a Case of Hereditary Ataxia, Brain **20**:276, 1897.

TABLE 4.—*Muscular and Motor Status (Forty-Six Cases)*

Author	Ataxia	Speech Defect	Difficulty of Swallowing	Atrophy and Asthenia	Back Deformity	Foot Deformity	Incontinence	Abnormal Muscle Movements
André-Thomas	Upper and lower limbs	Slow and nasal	.....	Lower limbs contracted	.....	Present	.....	.....
Auscher [9]	Upper and lower limbs	Difficult	.....	.....	Scoliosis	.....	.....	Choreiform
Barker, case 1	Upper and lower limbs	Difficult	Present	Extreme atrophy at end	.....	.....	None	Choreiform
Barker, case 2	Upper and lower limbs	Difficult	.....	No atrophy	.....	.....	None	.....
Barrett	Upper and lower limbs	Mute	Very marked	Atrophy, hypotonus	Scoliosis	Present	None	.....
Bloeq and Marinesco	Upper and lower limbs	Defective	.....	.....	Scoliosis	.....	Bladder	Oscillations of head
Dejerine and André-Thomas	Upper and lower limbs	Explosive	Present	Hypotonus	Scoliosis	Present	Bowel	Oscillations of head
Fergus, case 1	Upper and lower limbs	Staccato	.....	None	Scoliosis	Present	.....	Oscillations of head and neck
Fergus, case 2	Upper and lower limbs	Marked slurring	.....	Slight	None	Present	.....	Jerky movements of head
Hess	Upper and lower limbs, especially lower	Scanning	.....	Muscular relaxation	Scoliosis	Present	.....	.....
Hoffmann, case 1	Upper and lower limbs	.....	.....	None	Kyphoscoliosis	Present	.....	Oscillations of head
Hoffmann, case 2 [10]	Upper and lower limbs	Slow and indistinct	Present	Atrophy	Kyphoscoliosis	Present	.....	Grimaces
Hunt, case 1	Upper and lawer limbs	Jerky	.....	Slight	Scoliosis	Present	None	None
Hunt, case 2	Upper and lower limbs	Scanning	.....	Slight	Scoliosis	Present	None	Choreiform
Hunt, case 3	Upper and lower limbs	Defective	.....	None	Scoliosis	Present	None	Choreiform
Hunt, case 4	Lower limbs	.....	.....	None	None	Slight	.....	None
Lambrior [11]	Upper and lower limbs	Slow and nasal	.....	Lower limbs weak	Kyphoscoliosis	Present	.....	Oscillations of head and trunk
Letulle and Vaquez	Upper and lower limbs and face	Slow and scanning	None	Underdeveloped lower limbs	Scoliosis	None	.....	Oscillations of head
Lhermitte and Artom	Marked in lower limbs, slight in upper	None	.....	Hypotonus of lower limbs	Kyphoscoliosis	.....	Bladder	Epileptic crises
Litchfield, Latham and Campbell, case 1	Lower limbs	Late defect	.....	Slight hypotonus	None	Present	.....	.....
Litchfield, Latham and Campbell, case 2	Upper and lower limbs	Defective	.....	.....	.....	.....	.....	Jerking of head

Lloyd and Newcomer [1]	Lower limbs	Staccato	.....	No atrophy	Scoliosis	None	.....	.....
Mackay [6]	Present	Labored and explosive	.....	.....	Scoliosis	.....	.....	.....
Menzel	Upper and lower limbs	Difficult and jerky	Present	Progressive emaciation	.....	Present	None	Choreiform movements
Meyer [3]	Upper and lower limbs	Slow	.....	.....	.....	.....	.....	.....
Mott	Upper and lower limbs	Defective	.....	Asthenia	Scoliosis	Present	None	.....
Müller, E., case 1	Upper and lower limbs	Indistinct	Present	.....	Kyphoscoliosis	None	.....	Choreiform
Müller, E., case 2	Marked of lower limbs	Slow	.....	None	Kyphoscoliosis	None	.....	Athetoid and choreiform
Müller, W.[2]	Marked	Scanning	Present	Asthenia	.....	.....	Bowel	.....
Osnato, case 1	Upper and lower limbs	Defective	.....	Marked	Scoliosis	Present	.....	Choreiform
Osnato, case 2	Upper and lower limbs	.....	.....	Slight	.....	Present	.....	Choreiform
Osnato, case 3	Upper and lower limbs	Slight	.....	None	Scoliosis	None	.....	Choreiform
Osnato, case 4	Upper and lower limbs	None	.....	None	Scoliosis	Present	.....	Choreiform
Osnato, case 6	Upper and lower limbs	Defective	.....	Marked	Scoliosis	Present	.....	.....
Osnato, case 7	Upper and lower limbs	Defective	.....	Marked	.....	Present	.....	.....
Pfeiffer	Upper and lower limbs	Slurring	.....	Asthenia and emaciation	Scoliosis	Present	.....	Jerking of head and twitching of face
Philippe and Oberthür, case 1	Upper and lower limbs	Marked defect	.....	.....	Scoliosis	Present	.....	Oscillations of head
Philippe and Oberthür, case 2	Lower limbs only	Slow and jerky	.....	Atrophy	None	Present	.....	Oscillations of whole body
Pic and Bonnamour	Upper and lower limbs	Explosive	Difficulty	Asthenia, no atrophy	.....	Present	Bladder	Oscillations of head
Purser	Lower limbs	None	None	None	.....	Marked	.....	.....
Rennie [5]	Lower limbs	Slow and jerky	.....	None	Stooping	.....	.....	.....
Rütimeyer, case 1	Upper and lower limbs	Indistinct	None	Marked atrophy of lower limbs	Kyphoscoliosis	.....	.....	.....
Rütimeyer, case 2	Upper and lower limbs	.....	.....	.....	Kyphoscoliosis	.....	.....	.....
Simon [8]	Lower limbs	Slight	.....	.....	None	.....	Bladder	.....
Spiller [4]	Upper and lower limbs	Slow	.....	Atrophy of hands and feet	.....	Present	.....	.....
Thomas	Upper and lower limbs	Jerky and indistinct	.....	None	Slight scoliosis	Present	.....	.....

diseases. The longest duration of a case here recorded was one in which the patient survived thirty-seven years after the onset of the disease;[4] the shortest, one and a half years.[5] In twenty-four cases in which the length of life after the beginning of the condition was recorded, only five of the patients survived for more than twenty years.

Eye Symptoms.—Nystagmus is the most frequent eye symptom. It was present in 70 per cent of the cases recorded in table 2. The Argyll Robertson pupil was noted in three cases; ptosis, in four; optic atrophy, in two, and ophthalmoplegia, in one. Nystagmus, optic atrophy and ophthalmoplegia were present in the case that I am reporting. Nystagmus was more frequently noted in the cases of longer duration, suggesting that this is a symptom of relatively late appearance.

Reflexes.—The knee reflexes were absent in thirty-three of forty-four cases; they were weak in two. Active knee reflexes were encountered in eight of the forty-four cases; they were normal in one. The Achilles reflex, in general, was in accord with the knee reflex.

The Babinski reflex was present in 43 per cent of the cases in which the present or absence of this symptom was definitely stated.

Muscular and Motor Status.—In table 4 there are recorded forty-six cases in which there was a record of the presence or absence of ataxia, speech defect, difficulty of swallowing, atrophy and asthenia, deformities of the back and of the foot, incontinence of bladder or bowel and abnormal muscle movements.

Ataxia was present in every case; in the great majority, it involved all four extremities.

A speech defect was present in thirty-nine of the forty-two cases in which its presence or absence was recorded. Various adjectives, such as scanning, slurring, explosive, hesitating, slow, jerky, indistinct, etc., were used by different writers to describe the defect.

Difficulty in swallowing was recorded in eight cases. It is hard to appraise the importance of weakness and atrophy, as these symptoms must undoubtedly have been present in many of the cases in which they were not recorded.

Scoliosis was present in 83 per cent of the cases in which its presence or absence was recorded; deformities of the foot, in the same proportion. Incontinence was an unusual symptom; vesicular incontinence was recorded in only four cases, and rectal in two.

Various abnormal muscular movements were described most commonly as choreiform movements or as oscillations of the head.

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4. Spiller, W. G.: Friedreich's Ataxia, J. Nerv. & Ment. Dis. **37**:411 (July) 1910.

5. Rennie, G. E.: A Case of Friedreich's Hereditary Ataxia with Necropsy, Brit. M. J. **2**:129 (July 15) 1899.

Sensory Disturbances.—The observations in the cases reported with reference to sensation, especially sensibility to pain, temperature and touch, are shown in table 5. It would seem, from a study of this table, that pronounced sensory symptoms are not of common occurrence.

TABLE 5.—*Sensory Disturbances with Especial Reference to Pain, Temperature and Touch (Forty-Four Cases)*

Author	Sensory Disturbances
André-Thomas and Durupt	Pressure and bone sense impaired late in disease
Auscher [9]	Lightning pains
Barker, case 1	No disturbance
Barker, case 2	No disturbance
Barrett	No disturbance
Blocq and Marinesco	Left hemianesthesia from head to knee; taste and smell lost
Dejerine and André-Thomas	No disturbance
Fergus, case 1	Tactile and pain sense impaired
Fergus, case 2	No disturbance
Hess	No disturbance
Hoffmann, case 1	Pains in head and lower limbs
Hoffmann, case 2 [10]	No disturbance
Hunt, case 1	Definite impairment of tactile sense
Hunt, case 2	No disturbance
Hunt, case 3	No disturbance
Lambrior [11]	No disturbance
Letulle and Vaquez	No disturbance
Lhermitte and Artom	Pains in legs; total anesthesia of foot and leg to patella
Litchfield, Latham and Campbell, case 1	No disturbance
Litchfield, Latham and Campbell, case 2	No disturbance
Lloyd and Newcomer [1]	No disturbance
Mackay [6]	No disturbance
Menzel	Sense of pressure at base of skull and right shoulder
Meyer [3]	No disturbance
Mott	No disturbance
Müller, E., case 1	Tactile and deep sensibility of the arms and legs impaired
Müller, E., case 2	Slight impairment of deep sensibility
Müller, W.[2]	No disturbance
Osnato, case 1	Loss of position sense in toes
Osnato, case 2	No disturbance
Osnato, case 3	Slight changes in feet
Osnato, case 4	No definite changes
Osnato, case 6	Definite impairment of pain, thermal, tactile and deep sensibility
Osnato, case 7	Distinct hyperesthesia to pain, heat and cold
Pfeiffer	Slight tactile loss in lower limbs; muscle, vibratory and position sense diminished in hands and feet
Philippe and Oberthür, case 1	Normal except for shooting pains in knees and feet
Philippe and Oberthür, case 2	Paresthesias in lumbar region and calves
Pic and Bonnemour	Complete loss of sense of position
Rennie [5]	No disturbance
Rütimeyer, case 1	Decreased tactile sense in lower limbs
Rütimeyer, case 2	Tactile sense slightly impaired in arms and legs
Simon [5]	Sensation of pricking and intense heat in legs
Spiller [4]	Slight loss of tactile sense
Thomas	No disturbance

TABLE 6.—*Percentage Incidence of the Most Important Signs in the Tabulated Cases*

	Per Cent
Ataxia	100
Speech defect	93
Foot deformity	83
Scoliosis	83
Absent or weak knee and ankle reflexes	80
Nystagmus	70
Babinski reflexes	43

TABLE 7.—*Spinal Cord Observations at Necropsy (Eighteen Cases)*

Author	Posterior Column	Lateral Column	Anterior Column	Anterior and Posterior Horns	Size of Cord; Condition of Posterior Roots
André-Thomas and Durupt	Marked degeneration; Lissauer's tract contained healthy fibers	Crossed pyramidal, direct and indirect cerebellar tracts involved, but less at higher levels	Sclerosis decreasing at higher levels	Atrophy of posterior horn; Clarke's column atrophied	.....
Auscher [9].....	Sclerosed, Goll's tract more than Burdach's; glial fibers undulating; "tourbillons"; Lissauer's zone intact	Anterolateral column normal	Intact.....	Atrophy of posterior horns	Cord very small; posterior roots apparently normal
Dejerine and André-Thomas	Sclerosis, especially of Goll's tract; ventrolateral portion and Lissauer's preserved; "tourbillons"	Crossed pyramidal, direct and indirect cerebellar tracts slightly involved	.....	Posterior horn slightly atrophied; Clarke's column with few cells	Cord small; posterior roots sclerosed
Hoffmann [10].....	Marked degeneration, increasing in Goll's tract as top of cord is approached; less in Lissauer's	Lateral pyramids and cerebellar tracts degenerated	....	Clarke's column sclerosed, with almost complete absence of cells	Cord small; posterior root partly degenerated
Lambrior [11].....	Sclerosis from sacral region to medulla, particularly of Goll's tract	Sclerosis of crossed pyramidal, direct and indirect cerebellar tracts	.....	Posterior horns atrophied; Clarke's column sclerosed	Cord small; posterior roots atrophied
Letulle and Vaquez....	Extreme sclerosis of Goll's tract and less of Burdach's; Lissauer's tract sclerosed	Slight degeneration of crossed pyramidal tracts; moderate of direct cerebellar	Intact.....	Marked lesion of Clarke's column	Cord very small; posterior roots atrophied
Lhermitte and Artom.	Degeneration very marked in sacral, dorsal and lumbar regions; less in cervical; cornucommissural tract preserved; Lissauer's degenerated slightly	Crossed pyramidal tract intact	Peripheral portion degenerated, not involving Türck's tract	Both horns atrophied; cells not degenerated; Clarke's column sclerosed	Cord very small; posterior roots degenerated
Lloyd and Newcomer [1]	Sclerosed, especially in central area, the ventral and dorsal parts having healthy fibers	Crossed pyramidal and direct cerebellar tracts involved	Intact.....	Clarke's cells decreased and degenerated; anterior horn cells degenerated	Cord not small; posterior roots atrophied

Müller, E.	Sclerosis, marked in Goll's tract, less in Burdach's tract; ventrolateral and Lissauer's tract preserved; wavy arched glia fibers	Sclerosis of lateral pyramidal, direct cerebellar and part of indirect cerebellar tracts	Intact.....	Clarke's column sclerosed....	Cord small; posterior roots normal
Müller, E.	Marked degeneration, especially of Goll's tract; ventrolateral portion and Lissauer's tract preserved; peculiar wavy glia fibers; "tourbillons"	Very slight degeneration.....	.....	Clarke's column almost completely degenerated	Cord small; posterior roots almost normal
Pfeiffer.....	Sclerosis, most intense in middorsal region; Goll's tract in particular; cornucommissural fibers preserved	Crossed pyramidal, direct and indirect cerebellar tracts affected; indirect cerebellar tracts less involved	Intact.....	Clarke's cells and column degenerated; anterior horn cells show chronic degeneration	Posterior roots atrophied
Philippe and Oberthür (case 1)	Degeneration of Goll's tract marked; less of Burdach's	Sclerosis of crossed pyramidal, direct and indirect cerebellar tracts	Degeneration from medulla to upper part of dorsal region	Anterior horn normal; Clarke's cells and column sclerosed	Cord very small; posterior roots sclerosed
Philippe and Oberthür (case 2)	Marked degeneration in sacral and lumbar regions; cornucommissural preserved; Lissauer's tract poor in fibers	Degeneration of lower part of pyramidal tract	Marked sclerosis of Türck's zone, but less advanced than in posterior column	Posterior horns atrophied....	Cord flat and thin; posterior roots degenerated
Pic and Bonnamour...	Degeneration most marked in Goll's tract; Lissauer's tract also sclerosed	Complete sclerosis of crossed pyramidal and direct cerebellar tracts	Intact.....	Anterior horn normal; posterior horn atrophied; slight sclerosis of Clarke's column	Cord small; posterior roots degenerated
Rennie [5].....	Sclerosis, most complete in middorsal region; cornucommissural group preserved	Slight degeneration of crossed pyramidal and direct cerebellar tracts	Intact.....	.....	Cord not small
Richardson [*].....	Marked sclerosis; cornucommissural fibers preserved	Crossed pyramidal and direct cerebellar tracts involved	Affected on one side	.....	Cord small; posterior roots atrophied
Simon [3].....	Marked sclerosis, maximal in dorsal region	Crossed pyramidal and direct cerebellar tracts sclerosed	Direct pyramidal tracts partly affected	Clarke's column degenerated; cells of anterior horn diminished	Cord small; posterior roots sclerosed
Winkelman and Eckel†	Marked sclerosis, especially in lower levels; Goll's tract more than Burdach's tract	Crossed pyramidal, direct and indirect cerebellar tracts involved	Intact.....	Clarke's cells decreased; anterior horn cells show chronic degeneration	Cord small; posterior roots atrophied, especially at lower levels

[*] J. Boston Soc. Sc. **3**: 25, 1898.
† Arch. Neurol. & Psychiat. **13**: 37 (Jan.) 1925.

Pathologic Changes of the Spinal Cord.—In the eighteen cases in which lesions of the spinal cord were recorded (table 7), the pathologic changes were fairly uniform. They resembled, in most respects, those reported by Mackay [6] and arranged in a similar table. In fourteen cases of our group, the spinal cord was unusually small, and the posterior roots were degenerated. The three cases in which the cord was of normal size were of one and a half, three and eleven years' standing, respectively.

Sclerosis was most marked in the posterior columns, and was more intense in Goll's than in Burdach's tract; it diminished as the medulla oblongata was approached. The cornucommissural fibers were frequently well preserved. Lissauer's zone was sometimes severely injured; in other cases, it was intact.

The lateral columns were less involved than the dorsal region. The crossed pyramidal and the direct cerebellar tracts suffered the most. Seldom was involvement of the indirect cerebellar tract reported. In half of the cases, the anterior column was found intact; in five of the remaining nine, it was reported as more or less affected.

In the gray matter of the cord, the posterior horns were more frequently affected than the anterior. Clarke's cells were almost invariably reported as diminished or degenerated. In a few cases, there was partial destruction of the anterior horn cells.

The glial replacement was described by a number of the authors as being in whorls ("tourbillons") of wavy hairlike fibers surrounding minute blood vessels. This fiber arrangement is considered to have an important significance by Bramwell,[7] Simon,[8] and others; Mackay,[6] on the contrary, denies its importance.

There is evidence to show that the extent of the cord lesions depends, to a large degree, on the duration of the disease. This, however, is not always the case. In some cases of relatively short duration, for example, those of Auscher,[9] Hoffmann,[10] and Lambrior,[11] the destruction was almost as great as in those of much longer standing.

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6. Mackay, H.: Pathology of a Case of Friedreich's Disease, Brain **21**: 435, 1898.

7. Bramwell, B.: Notes of Three Cases of Friedreich's Disease, Atlas of Clinical Medicine, Edinburgh, 1891-1892, vol. 1, p. 178.

8. Simon, J.: Un cas de maladie de Friedreich avec autopsie et examen histologique, Progrès méd. **6**:145 (Sept. 4) 1897.

9. Auscher, E.: Sur un cas de maladie de Friedreich (sclérose névroglique pure), suivi d'autopsie (étude histologique de la moelle epinière et des nerfs cutanés), Compt. rend. Soc. de biol. **2**:475, 1890.

10. Hoffmann, J.: Pyramidenseitenstrang-Symptome bei der hereditären Friedreichschen Ataxie; Sektionsbefund., Deutsche Ztschr. f. Nervenh. **60**:179 (Aug. 17) 1918.

11. Lambrior, A. A.: Un nouveau cas de maladie de Friedreich avec autopsie, Rev. neurol. **25**:57 (Jan. 30) 1913.

CONCLUSIONS

It may be seen from the accompanying tables that ataxia of both lower and upper extremities, speech defect, scoliosis, foot deformities, absent or weak knee and ankle reflexes and nystagmus are the most constant and frequent symptoms of Friedreich's ataxia. However, the ataxia may be confined to the lower extremities, and the knee reflexes may be exaggerated. The type of speech defect, the presence of nystagmus and the deformities of the foot or back may show considerable variation.

Among the symptoms of less common occurrence are the Babinski reflex, abnormal muscular movements, muscular atrophy and hypotonus, pupillary disturbances, optic atrophy, ptosis of the eyelids and ophthalmoplegia.

The resemblance between Friedreich's ataxia and tabes dorsalis is suggested not only by the pathologic changes in the spinal cord in the two diseases, but also by the number of clinical symptoms that are common to both. The occasional presence in Friedreich's ataxia of Argyll Robertson pupils, optic atrophy, bladder and bowel incontinence and muscular hypotonus combines with the ataxia that is so characteristic of both diseases to form the clinical parallelism.

It may be assumed that the clinical manifestations serve as a fair index of the stage of the organic changes in the central nervous system. At times, however, this correlation is absent; for example, it is not a rare occurrence to find marked degenerative changes in the pyramidal tracts in cases in which, during life, the Babinski reflex was never elicited.

With reference to the two cases that I am reporting for comparison, it is a fair supposition that the pathologic condition of the younger patient, three years after the onset of the illness, was much the same as that of his brother at the time of the latter's death. Certainly the clinical signs were almost similar at this period. The younger boy survived four years longer, during which time the clinical symptoms made a fairly orderly advance. In the case of shorter duration, the degenerative changes in the spinal cord were limited to a certain area of the posterior columns; in the case of longer duration, spinal cord destruction was much more extensive. In neither case were the anterior columns involved; in both, the degeneration of the lateral columns remained stationary.

SOMNOLENCE:

ITS OCCURRENCE AND SIGNIFICANCE IN CEREBRAL NEOPLASMS *

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Somnolence as a symptom in cerebral lesions has been of particular interest since the recent experience with epidemic encephalitis emphasized its incidence and importance. The vast amount of literature dealing with the latter subject does not lead to definite conclusions as to the cause of somnolence, nor does the scanty literature on its occurrence in expanding lesions give more than vague and sometimes misleading information on the subject. It has been a matter of general experience to meet diagnostic problems in which somnolence has given rise to confusion, when other cerebral symptoms, such as headache, cranial nerve palsies and slight changes in the optic nerve heads, suggested epidemic encephalitis, neoplasm, meningitis and so on. Difficulties in several cases led us to investigate the subject from the standpoint of expanding intracranial neoplasms, in an attempt to understand the physical causes underlying the production of somnolence.

The occurrence of hypersomnia has long been noted in brain tumors. Hercouet,[1] in 1905, concluded from a study of six of his own cases and a number of those reported by other observers that the symptom had no localizing value; that it occurred most frequently in tumors near the base of the brain, and that the type of tumor growth was more frequently sarcoma. He suggested that there might be a sleep center in the region of the pituitary body which is involved in cases of brain tumor in which somnolence is a predominating feature. In Hoppe's case,[2] somnolence was noted particularly in a tumor of the corpora quadrigemina, in which the Sylvian aqueduct was obliterated, with tumor extension into the substance of the pons.

Goodhart and Climenko[3] reported, in 1915, one of seven cases of brain tumor in which hypersomnia was a conspicuous symptom. The growth implicated the optic chiasm, optic nerves, olfactory lobes and

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* Read at the Fifty-Second Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1926.

1. Hercouet, J.: Le sommeil dans les tumeurs cérébrales, Paris Thesis, 1905.

2. Hoppe, H. E.: A Tumor of the Corpora Quadrigemina, J. Nerv. & Ment. Dis. **39**:108, 1912.

3. Goodhart, S. P., and Climenko, H.: Seven Cases of Brain Tumor with Special Reference to Differential Diagnosis, J. Nerv. & Ment. Dis. **42**:193, 1915.

tracts, pituitary body, frontal lobes on each side, the corona radiata on each side, and the anterior portion of the corpus callosum. Climenko,[4] in 1916, described a case in which "yawning and drowsiness" were frequent manifestations together with diminished intelligence. A glioma of the white matter of the right cerebral hemisphere was found on operation. In 1919, Vernie [5] reviewed two cases of tumor of the third ventricle. Profound somnolence, from which the patient could not be aroused for several hours, occurred in one, and in the other repeated attacks of somnolence were especially prominent becoming more profound and more prolonged in the last four months of life.

In 1920, Foster Kennedy [6] noted drowsiness and frequent yawning with a lowering of mental acuity in patients with ventricular distention. He emphasized the symptom in frontal lobe tumors. In tumors of the corpora quadrigemina he believed the intense drowsiness was due to the great intracranial pressure that existed. He believed, however, that morbid sleep might well be a focal symptom of subthalamic disease quite apart from general pressure conditions produced by the tumor growth. In 1920, Lenoble [7] and his associates reported a case in which somnolence was a prominent feature from the onset of symptoms. Postmortem examination revealed a tumor of the parietal region. In 1921, Christiansen [8] noted four cases of brain tumor, one supratentorial and three infratentorial in location, in which "torpor" was a prominent symptom. Morrison,[9] in 1922, described a case of melanoma of both frontal lobes, with nodules in the left parietal and occipital lobes in which the patient was "quite drowsy" at times.

In 1923, Claude [10] and his associates reported a case of increasing somnolence in which the patient slept "all the time" for two weeks. A gradual clearing of the somnolence was followed by diplopia and headache, and symptoms suggestive of Parkinson's disease. Necropsy revealed a large glioma in the right cerebral hemisphere invading the lenticular nucleus and extending into the brain stem. In 1923,

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4. Climenko, H.: The Diagnosis of Brain Tumor, New York M. J. **103**:305 (Feb. 12) 1916.

5. Vernie, L.: L'étude du syndrome infundibulaire dans les tumeurs du troisième ventricale, Paris Thesis, 1919.

6. Kennedy, Foster: Tumors of the Intracranial Cavity, in Nelson: Loose-Leaf Living Medicine **6**:119, 1920.

7. Lenoble, E. Baumier, and Dujardin: Brain Tumors Suggesting Encephalitis, Bull. et Mém. Soc. méd. d. hôp. de Paris **44**:1577 (Dec. 17) 1920.

8. Christiansen, V.: Les tumeurs du cerveau, Paris, Masson & Cie., 1921.

9. Morrison, A. W.: Case of Melanoma of Brain Resembling Epidemic Encephalitis, Journal-Lancet **42**:37 (Jan. 15) 1922.

10. Claude, H.; Schaeffer, H., and Alajouanine, T.: Brain Tumor Simulating Encephalitis, Paris méd. **13**:337 (April 14) 1923.

Cushing [11] reported an extensive series of intracranial tumors in which two cases of pituitary adenoma presented excessive drowsiness.

In 1925, Globus and Strauss [12] reported sixteen cases of spongioblastoma multiforme, and described six in which the diagnosis of epidemic encephalitis was suggested. In five of these cases drowsiness was present in a more or less marked degree. The location in these instances was: right frontoparietotemporal; brain stem and pontocerebellar angles: right temporal lobe; right frontoparietal region and right side of corpus callosum; left cerebral hemisphere.

The literature for the most part, then, gives no definite information as to the location or type of tumors in which hypersomnia occurs, and the physical modus operandi is in no wise explained.

In our study of this problem we have taken one hundred consecutive cases of intracranial tumor, verified by operation or at postmortem examination. Many cases in which the diagnosis was established beyond reasonable doubt by the state of existing increased tension and by probing were not included in the series because the tumors were not actually observed. Several cases had to be excluded in which somnolence was a most marked feature, in undoubtedly deeply situated tumors, which were not observed directly or by postmortem examination.

Whenever the term somnolence is used in the course of this discussion it refers to definite hypersomnia, which was in no case due to medication, insufficient sleep during the previous night, exhaustion from pain, convulsive episodes, operative shock, febrile states, terminal stages or chance somnolence. In each case notes were made by the attending physician, house officer or nurse in the daily progress notes.

The type of somnolence is noted as early or late in the known existence of symptoms. The degree is indicated as slight, moderate or marked. By slight is meant repeated recurrence of drowsy periods from which the patient aroused spontaneously. By the term moderate is meant more prolonged periods from which it was often necessary to arouse the patient. By marked somnolence we refer to repeated attacks of stuporous states from which it was often difficult to arouse the patient, and by a return to the somnolent state after awakening.

Somnolence was present in thirty-two of the 100 cases of this series. It was noted as marked in twenty of these, in ten of which it occurred early (within the first few months) and in the remaining ten late in the course of the disease. Moderate drowsiness was noted in six cases, in two of which it occurred early. Slight somnolence occurred in six cases, late in the disease and inconstant in type. Drowsiness was the first

11. Cushing, Harvey: Notes on a Series of Intracranial Tumors, Arch. Neurol. & Psychiat. **10**:605 (Dec.) 1923.

12. Globus, J. H., and Strauss, Israel: Spongioblastoma Multiforme, Arch. Neurol. & Psychiat. **14**:139 (Aug.) 1925.

symptom noted in four cases. It was constantly present in seventeen, and early in eight of these. It was an early symptom in twelve, and late in twenty.

The age and sex of the patient bore no significance as to the time of onset, type or degree of somnolence. In three cases in which somnolence was a conspicuous feature headache and vomiting were absent.

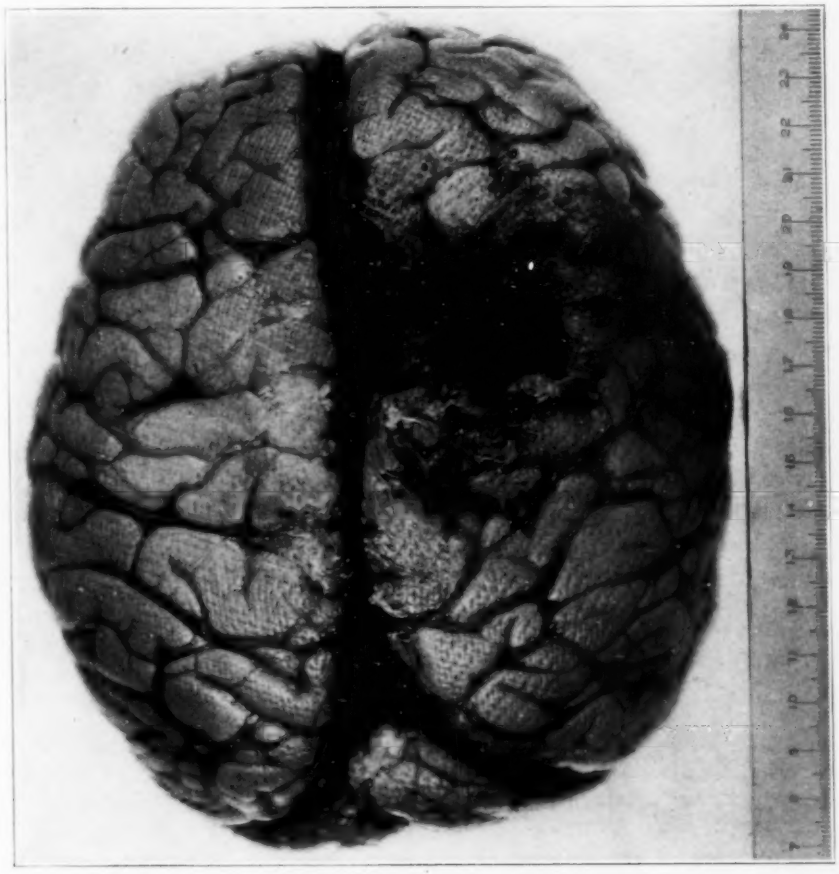

Fig. 1.—Endothelioma of right frontoparietal region; ventricles normal; somnolence late, constant and marked.

Concerning the location of the neoplasms: the cerebellum was involved in nine cases; the frontal lobes in eight (seven of them right frontal); the midbrain in two; the pituitary body in two; the temporal lobes in two; the parietal lobes in three; the parieto-occipital lobes in three; the temporoparietal lobes in two, and the frontoparietal lobes in one.

The location and type of tumor in which somnolence was early, persistent and marked were as follows: (1) left frontal—glioma; (2) right frontal—glioma; (3) pituitary body—adenoma; (4) right temporal—glioma; (5) left parietal—glioma; (6) right fronto-parietal—endothelioma; (7) right and left optic thalamus and third ventricle—type doubtful; (8) left parieto-occipital—glioma; (9) cerebellum—glioma; (10) left temporo-occipital—fibroblastoma.

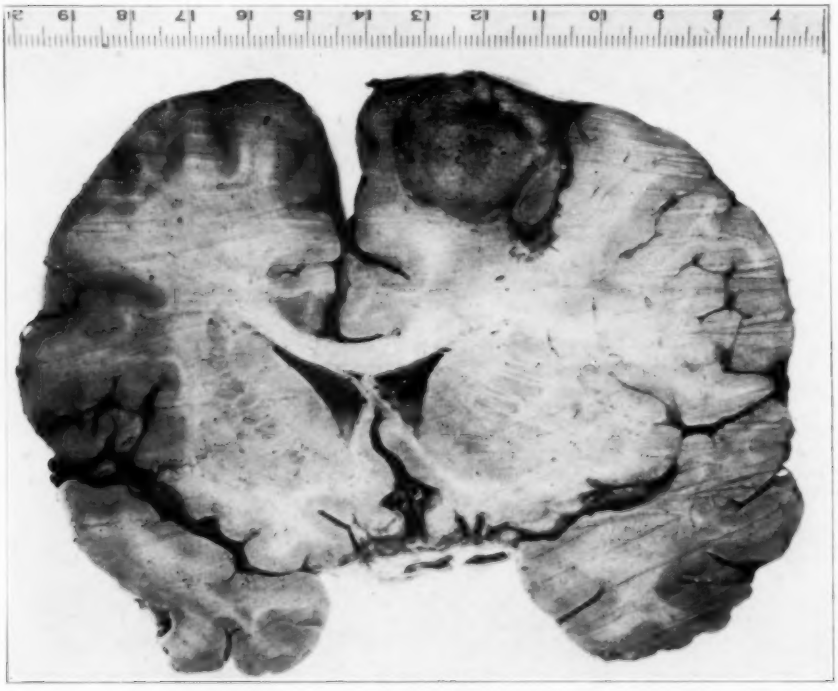

Fig. 2.—Pearly endothelioma of right frontoparietal region; no hydrocephalus, moderate hyperplasia of right cerebral hemisphere; somnolence early (three months after first symptom), constant and marked.

The types of tumors encountered were: endothelioma, 8; sarcoma, 1; adenoma, 1; glioma with cystic degeneration, 6; glioma without cyst, 14; undetermined, 2.

At attempt has been made to determine whether distention of the ventricles has played any significant part in the production of somnolence. In certain instances we have been forced to rely entirely on the ventricular pressure as determined by surgical tapping.

In ten cases, postmortem examination has given incontrovertible information. In twenty-three cases, the information derived from either source is considered conclusive. In fifteen, there was internal hydrocephalus

of varying degree. In eight, the ventricles were normal. In nine cases of marked ventricular dilatation the somnolence was, with two exceptions, late in occurrence, and varied in degree from slight to marked; and in eight cases in which there was no ventricular distention, som-

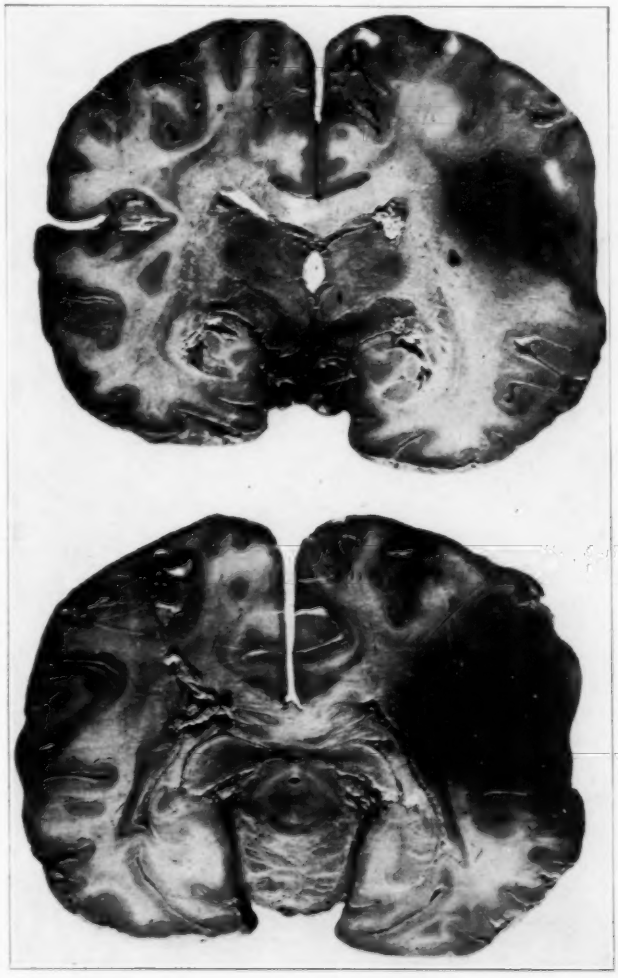

Fig. 3.—Glioma of right parietal lobe; no dilatation of lateral ventricles, slight dilatation of third ventricle; no hyperplasia of brain; somnolence late, inconstant and marked.

nolence was an early symptom in three, a marked factor in six, and in three of these cases it was constantly present. In six cases—in which slight internal hydrocephalus was found—somnolence was a marked feature in three instances and a constant symptom in four.

Status of the Thirty-Two Cases Manifesting Somnolence

Case	Age and Sex*	Duration of Symptoms	First Symptom	Presenting Signs of Increased Intracranial Pressure	Ventricular Status	Location of Tumor	Hyperplasia of Region Involved	Type of Tumor and Size	Somnolence		
									Order of Appearance	Type	Degree
1	5, ♀	9 mo.	Headache	Headache; diminished vision	.....	Left and right cerebellar hemispheres and vermis	.....	Cystic glioma	Late	Inconstant	Slight
2	24, ♀	10 mo.	Headache	Headache; vomiting; papilledema, 3 degrees	No increase of tension	Right frontal lobe	.....	Endothelioma, 8 × 12 cm.	Late	Inconstant	Slight
3	40, ♀	4 mo.	Headache	Headache; papilledema, 3 degrees	Marked dilatation of all ventricles except fourth, including aqueduct, postmortem	Cerebellar hemispheres, vermis and peduncles; extension into fourth ventricle	.....	Sarcoma	Late	Inconstant	Slight
4	45, ♂	3 mo.	Headache	Headache; vomiting	Normal ventricles postmortem	Right parietal lobe	None	Glioma, 4 × 3 cm.	Late	Inconstant	Marked
5	15, ♀	?	Headache	Headache; papilledema, 2 degrees	Unsuccessful tapping	Left parieto-occipital	.....	Glioma, 6 × 8 cm.	Late	Inconstant	Slight
6	37, ♂	6 mo.	Headache	Headache; dizziness; papilledema, 4 to 7 degrees	Moderate tension	Left cerebellar hemisphere	.....	Endothelioma, 12 × 8 cm.	Late	Inconstant	Slight
7	48, ♂	2 mo.	Headache	Headache; aphasia; vomiting; no papilledema	.....	Left parietal lobe	.....	Calcified glioma	Early	Constant	Marked
8	45, ♂	4 yr.	Drowsiness	Drowsiness; memory defects; convulsions; papilledema, 3 to 2 degrees; sella atrophy	.....	Left temporo-occipital	.....	Fibroblastoma, 6 × 5 × 5 cm.	Early	Constant	Marked
9	48, ♂	11 wk.	Convulsion	Headache; convulsions; papilledema, 3 degrees; uncinate fits	Slight distention of right ventricle, postmortem	Left parieto-occipital	Present	Glioma, 2.5 × 4 cm.	Early	Inconstant	Moderate
10	?, ♀	7 mo.	Weakness and incoordination left fingers	Hemianopia; memory defect; headache; drowsiness; sella absorption	.....	Right temporo-occipital	.....	Endothelioma, 6 × 5 cm.	Late	Constant	Marked
11	50, ♀	2 yr.	Weakness of left lower extremity	Left hemiplegia; papilledema	No increase of tension	Right precentral gyrus	.....	Endothelioma, partly cystic, 2 × 2.5 cm.	Late	Inconstant	Slight
12	41, ♀	3 yr.	Headache	Bitemporal hemianopia; diplopia; headache; drowsiness	.....	Pituitary	.....	Adenoma	Early	Constant	Marked
13	56, ♂	2 wk.	Drowsiness	Headache; drowsiness; papilledema 3 to 4 degrees	No increase of tension	Left frontal lobe	.....	Glioma, cystic, 2 × 2.5 cm.	Early; first symptom	Constant	Very marked

14	52, ♂	2 yr.	Personality change	Partial aphasia; no papilledema; normal roentgen ray	.....	Left parietal	.....	Glioma, cystic	Late	Constant	Marked
15	26, ♂	2 mo.	Petit mal	Sella atrophy	Marked increase of tension	Left cerebellar hemisphere	.....	Endothelioma	Late	Inconstant	Moderate
16	54, ♀	26 mo.	Petit mal	Headache; vomiting uncinate attacks; papilledema, 3 to 4 degrees	No increase of tension	Right temporal	.....	Glioma, cystic	Late	Constant	Marked
17	50 ♂	5 mo.	Weakness left upper and lower extremities	Headache; memory defect; diplopia; left hemiparesis drowsiness; papilledema; sella erosion	Normal ventricles postmortem	Right frontoparietal	Present	Pearly endothelioma, 2 × 2.5 cm.	Early	Constant	Marked
18	58, ♀	4 mo.	Headache	Headache; vomiting; papilledema; slight sella changes	Normal ventricles postmortem	Right frontal	Present	Glioma, 4 × 5 cm.	Late	Inconstant	Marked
19	3, ♀	1 yr.	Staggering gait	Strabismus; staggering; vomiting; papilledema	Marked increase of tension	Right cerebellar hemisphere	.....	Glioma, 3 × 4.5 cm.	Late	Inconstant	Moderate
20	38, ♀	5 mo.	Numbness right	Marked drowsiness; aroused with difficulty; headache; papilledema, 1 to 2 degrees; sella absorption	Marked bilateral hydrocephalus postmortem	Right and left optic thalami occluding third ventricle, invading midbrain and left side of pons	.....	Glioma, 5 × 2 cm.	Early	Constant	Marked
21	40, ♂	4 yr.	Headache; drowsiness	Headache; no papilledema	No increase in tension	Right frontal	.....	Glioma with degeneration	Early	Inconstant	Marked
22	27, ♂	2 mo.	Ataxia	Headache; staggering; slight papilledema; slight sella absorption	Moderate increase of tension	Right and left cerebellar hemispheres	.....	Glioma	Early	Constant	Marked
23	3, ♀	3 mo.	Drowsiness; tilting of head	General paralysis of both lower extremities and right upper extremity; marked papilledema; drowsiness	Marked increase of tension	Right and left cerebellar hemispheres	.....	Cystic glioma	Early	Constant	Marked
24	5, ♀	11 mo.	Headache; vomiting	Headache; vomiting; increasing size of head; papilledema, 3 degrees; sella atrophy	Marked increase of tension	Right cerebellar hemisphere	.....	Cystic glioma	Late	Constant	Moderate to marked
25	37, ♂	1 mo.	Jacksonian attacks	Left hemiplegia; slight papilledema	.....	Right frontal	.....	Glioma	Late, following decompression	Constant	Marked

* In this column, ♂ indicates male; ♀, female.

Status of the Thirty-Two Cases Manifesting Somnolence—(Continued)

Case	Age and Sex	Duration of Symptoms	First Symptom	Presenting Signs of Increased Intracranial Pressure	Ventricular Status	Location of Tumor	Hyperplasia of Region Involved	Type of Tumor and Size	Somnolence		
									Order of Appearance	Type	Degree
26	10, ♂	3 yr.	Weakness of right lower extremity	Gait defect; headache; papilledema; drowsiness	Marked increase of tension	Right cerebellar hemisphere	.....	Glioma	Late	Constant	Marked
27	13, ♀	2 mo.	Nausea and vomiting	Vomiting; headache; papilledema, 5 degrees	Marked bilateral hydrocephalus with dilatation of third ventricle postmortem	Midbrain; invading also fourth ventricle	.....	Spongioblastoma, 3 × 2 cm.	Late	Constant	Marked
28	19, ♀	2 yr.	Strabismus	Optic atrophy; headache; drowsiness; sella destroyed	Moderate increase of tension	Pituitary and suprasellar region	.....	Strumous material	Early	Constant	Moderate
29	46, ♀	10 yr.	Headache	Headache; nausea; drowsiness; papilledema, 3 to 4 degrees; sella destroyed	Slight dilatation left ventricle postmortem	Right frontal lobe	Present	Glioma	Late	Constant	Marked
30	35, ♀	8 yr.	Convulsion	Amnesia; convulsions; left upper extremity general paralysis; papilledema	Ventricles normal postmortem	Right frontotemporal	.....	Endothelioma, 4 × 3 × 7 cm.	Late	Inconstant	Moderate
31	?, ♂	2 mo.	Headache	Apathy; mental confusion; staggering; headache; papilledema 3 to 4 degrees; drowsiness	Slight contralateral distention postmortem	Right temporal	None	Glioma, 5 × 6 cm.	Early, first week	Constant diurnal; restless at night	Marked
32	28, ♂	9 mo.	Headache	Headache; nausea; memory defect; right hemiplegia; speech disturbance; slight papilledema; sella absorption	No increase of tension; intracranial pressure very marked	Left parietooccipital	.....	Endothelioma, 10 × 12 cm.	Late	Inconstant	Moderate

In four cases examined postmortem, there was hyperplasia of varying degree of the lobe or lobes involved. In one of these, in which there was no ventricular distention, somnolence was constant, early and marked. In the second, likewise with no internal hydrocephalus, it was late, inconstant and marked (right frontal). In the third, with slight internal hydrocephalus, it was early, inconstant and moderate. In the fourth with slight ventricular distention, somnolence was late, constant and marked.

Normal sleep has been inadequately explained up to the present. That the circulation undergoes changes has been shown in man by phlethysmographic observations, and in lower animals by direct kymographic experiments. Mosso, in 1881, through a trephine opening, found that during sleep the volume of the brain diminished. "Unfortunately," as Howell [13] says, "this opinion was contradicted by Shepard" who claimed that during sleep there was a vascular dilatation in the brain as well as in the limbs. Howell concludes that after the onset of sleep the blood flow through the brain diminishes rapidly to a certain point and that, before awakening, the blood flow begins to increase again until it reaches normal proportions. His theory of normal sleep production is that the vasomotor center in the medulla loses its tone in consequence of fatigue from continued activity. Another factor was the greater or less fatigue of cortical cells bringing about a partial loss of acute consciousness.

Howell and Hill interpret sleep as due to cerebral anemia. Shepard finds the brain hyperemic and considers that the brain has an efficient supply of vasomotor nerves which during waking hours are in tonic activity, but in sleep suffer a diminution of tone leading to a local dilatation.

Hence we have two schools of thought directly at variance with each other, as far as the circulation of the brain during sleep itself is concerned.

In our studies of pathologic sleep we find that in expanding lesions of the brain somnolence is a frequent symptom, accompanied by signs of increased intracranial pressure not dependent on ventricular distention, in persons whose entire field of consciousness is variably disturbed. The increase of intracranial pressure undoubtedly affects the general cerebral circulation in such a way as to produce somnolence, and from our clinical studies we believe that a slowing of the blood supply is largely responsible for this phenomenon.

It is theoretically possible, at least, that, especially in tumors situated near the base of the brain, mechanical pressure on or narrowing of the vessels of the circle of Willis may result in a considerable slowing and a

13. Howell: Am. J. Physiol. **23,** 1909.

diminished volume of the cerebral blood supply. Varying degrees of pressure exerted on the Sylvian artery itself may, because of its extensive distribution, result in a considerable dulling of conscious reception of and response to stimuli that usually excite and maintain normal response and conscious activity.

On the other hand, it has been demonstrated that animals from which the cerebral hemispheres have been removed still maintain regular alternations of sleep and waking. While sleep, possibly, is not purely a cerebral phenomenon, there is little doubt that its intrinsic nature is concerned with the central nervous system.

The problem of the possibility of intoxication by metabolic products in neoplasms of the brain cannot be entered on here for want of scientific data.

The variety of the anatomic localizations of the neoplasms in our series, together with the approximate similarity of the resulting somnolence, leads us to believe that no discrete area of the brain exclusively controls the mechanism of sleep. We believe it is true that a diminution of cortical cell activity and a blunting of consciousness are intimately concerned in the production of sleep, and that these factors, together with slowing of cerebral circulation, cause somnolence.

CONCLUSIONS

1. Somnolence may occur in cerebral neoplasms regardless of the anatomic region involved.

2. It may occur before any definite signs of increased intracranial pressure manifest themselves.

3. It may occur without demonstrable gross changes in the ventricles.

4. It may occur without apparent gross hyperplasia of the region involved and without ventricular distention.

5. In our series somnolence was most constantly found in cases exhibiting marked internal hydrocephalus. On the other hand, the degree was often slight compared with other instances in which there was slight or no distention of the ventricles.

6. The majority of our cases showed indubitable signs of increased intracranial pressure, and it is our belief that this factor, with or without ventricular distention, operates directly or indirectly in slowing cerebral circulation, diminishing conscious receptivity of environmental stimuli and producing somnolence.

DISCUSSION

DR. HERMAN H. HOPPE, Cincinnati: In connection with somnolence, I was struck with Dr. McKendree's statement that frequently it is impossible to differentiate between the somnolence of encephalitis and that produced by great intracranial pressure. I would like to tell of a patient who had slept for nine months.

This woman was under treatment for changes of the menopause. She did not wake up spontaneously but could be aroused. Intelligence was normal except for a great slowing up. It took her a long time to answer questions. She was taken from the bed, led to the dining room where she ate her meals, but she had to be encouraged to swallow. There had never been any subjective signs of increased intracranial pressure. She had never had headaches, vomiting or vertigo, but the physical examination revealed enormous choked disks, widely dilated pupils which did not respond to light or in accommodation, and the variable symptoms of pressure would cause a Babinski reflex on one foot or a slight tendency to ankle clonus, which again would disappear.

Repeated spinal punctures did not relieve the somnolence, and the laboratory reports were invariably negative, but the spinal pressure was increased.

The diagnosis between encephalitis and neoplasm was impossible. She died nine months after the somnolence appeared, but permission for necropsy was not obtained.

DR. M. ALLEN STARR, New York: I recall a case that may be of interest in this connection. A woman who was suffering from some of the symptoms of brain tumor manifested this somnolent condition more or less for six months before death. Necropsy showed a tumor of both frontal lobes involving the corpus callosum. There was no choked disk or marked headache.

DR. EDWARD W. TAYLOR, Boston: Have you drawn any deductions as to somnolence from the nature of the tumor, and has the peculiarly infiltrating character of the gliomas possibly something to do with it?

DR. JOSEPH H. GLOBUS, New York: I also would like to mention another case in support of what Dr. McKendree has brought out, in which necropsy was performed a year ago. A man entered the hospital twice for observation. On the first admission he presented a picture which was so strikingly similar to that of encephalitis that a diagnosis of acute epidemic encephalitis was made. It was only on the second admission that doubt was expressed and the question of neoplasm came up. The first diagnosis was at no time fully dismissed, for the patient presented disseminated features characteristic of encephalitis.

At necropsy, an extensive neoplasm and, what would explain the dissemination of signs, numerous hemorrhages in the midbrain and pons were found. Very likely these hemorrhages were responsible for the external ophthalmoplegia which of course confused the clinical picture.

It seems that the similarity between encephalitis and brain tumor with relation to somnolence is to be found in the slowing of circulation. Any one who has studied the brains in cases of acute epidemic encephalitis soon after removal will recall that all cerebral vessels are markedly congested.

DR. DONALD GREGG, Boston: If I had known that Dr. McKendree was studying these cases of somnolence, I would have been glad to give him a report of my experience with a woman aged 21, who had been under observation for about two and a half years at various institutions. Her condition had been diagnosed as dementia praecox and also manic-depressive insanity.

When convalescent at the hospital, she did not complain of headache or of difficulty with the eyes. She was interested in and possessed talent in interpretive dancing and showed no evidence of incoordination of muscles. When she was discharged from the hospital, the only residual symptoms were sleepiness in the morning and a feeling that something had happened to her personality that left her feeling different from what she had ever felt before. She

was sent to the country for further convalescence. She was reported as doing excellently except for sleepiness in the morning. She was driving an automobile.

Ten days after leaving the hospital, she complained of being so sleepy that it was suggested that she lie down in the hammock on the porch. She had had no other symptoms and had driven an automobile the day before. She slept all afternoon and could not be roused in the evening. She continued to sleep more and more heavily without rousing during the next day and night, and thirty-eight hours after lying down in the hammock, stopped breathing.

Necropsy was performed to determine the cause of death. The pathologic report was a glioma about the size of a walnut embedded in both lobes of the cerebellum.

DR. CHARLES A. MCKENDREE: Answering Dr. Taylor's question, I would say that we were unable to come to a definite conclusion as to any special type of tumor producing somnolence. We were not surprised to find a preponderance of gliomas in our series, as they are the most frequent tumors of the brain.

We are quite familiar with the work done by Doctors Globus and Strauss, in 1925. In five of their cases, the difficulties in differential diagnosis between epidemic encephalitis and tumor of the brain were considerable. The tumors in their cases were found in five different regions of the brain.

TUBERCULOMA OF THE BRAIN

ITS INCIDENCE AMONG INTRACRANIAL TUMORS AND ITS SURGICAL ASPECTS *

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To one who has seen only two instances of tuberculoma of the brain during two and one-half years' association with a clinic in which neurologic surgery for patients of all ages plays an important part, the traditional estimate that the majority of all intracranial growths are granulomas and approximately half of them tuberculomas seems like a myth.

Their apparent present-day rarity, in this country at least, is so striking when compared with the statements in textbooks and current literature that it warrants comment. Furthermore, the fact that the two patients referred to (cases 6 and 14) both succumbed ultimately to the disease has aroused curiosity as to whether the results of surgical intervention for tuberculomas of the brain have been as unfavorable in other hands as they have been in this clinic. Hence this study was undertaken a year ago at Dr. Cushing's suggestion, with a twofold purpose.

INCIDENCE

M. Allen Starr,[1] in 1890, published in Keating's "Cyclopaedia" a table of approximately 300 intracranial tumors occurring in persons under 19 years of age as they had been recorded in the current medical journals up to that time. Three years later, for purposes of comparison, he tabulated in his monograph on brain surgery [2] an equal number of tumors as they had been found in adults. The figures showed that among the tumors in patients under 19 there were 152 tuberculomas (50.8 per cent), whereas among the 300 in patients above that age there were 41 (13.6 per cent), making 32.2 per cent for all ages. The gliomas, which had the next highest percentage, represented only 15.2 per cent for all ages, a surprising calculation in view of the approximately

* From the Surgical Clinic of Dr. Harvey Cushing.

* Read by invitation at the Fifty-Second Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 1, 1926.

1. Starr, M. A.: Intracranial Tumors, in Cyclopaedia of Diseases of Children, Philadelphia, edited by J. M. Keating, 1890, vol. 4, pp. 551-589.

2. Starr, M. A.: Brain Surgery, New York, William Wood & Co., 1893, p. 202.

40 per cent of gliomas in the series of verified tumors listed in the Peter Bent Brigham Hospital records.[3]

Starr's estimates have become so firmly fixed in the literature that they have largely influenced the figures given by most subsequent writers. Thus Peterson,[4] in 1894, added thirty-five cases of tumor to Starr's original list; the tuberculomas in the 335 cases proved four times more numerous than the gliomas and represented 49.5 per cent of the series. Even in the recent literature of children's diseases, the belief is reiterated that tuberculous tumors are the most common of all intracranial growths, and represent more than half of all brain tumors in childhood. Ziehen,[5] in 1912, gave a personal estimate of 50 per cent and quoted Seidl as having given a figure as high as 86 per cent.

Consequently, there can be no doubt of the frequency of solitary tubercles of the brain in the past and the large rôle they may have played clinically—and may possibly still play in certain countries. The statistics given by pathologists are somewhat confusing, for different authors have presented the subject from different angles. However, it may be said briefly that the percentages of solitary tubercles in cases of intracranial tuberculosis appear to have varied from 11 per cent in certain German clinics to the surprisingly high figure of 28.9 per cent quoted in 1903 from the Leeds General Infirmary, where Trevelyan[6] found 33 examples among 114 necropsies disclosing tuberculosis of the central nervous system. Prof. M. J. Stewart of Leeds informs me that there has been no marked, if any, diminution in these percentages in the twenty years since Trevelyan's report.

For the sake of comparison with these figures, I have been permitted, through the courtesy of Prof. S. B. Wolbach, to examine the records of the Children's and Infants' Hospitals in Boston. In the ten year period since 1915, there have been ninety-eight necropsies for tuberculosis of the brain or meninges, solitary tubercles having been disclosed in only nine instances (9.1 per cent), all the patients having been under 12 years of age.

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3. Starr's tables, which were standard for their time, are somewhat difficult to interpret according to our present terms of classification. If the cysts, sarcomas and gliosarcomas had been included with the gliomas, they would represent 45.5 per cent of all tumors; but this would exclude the endotheliomas, acoustic neuromas and pituitary adenomas.

4. Peterson, F.: Tumors of Brain and Meninges, in American Textbook of Diseases of Children, Philadelphia, edited by L. Starr, 1894, pp. 636-646.

5. Ziehen, T.: Die Krankheiten des Gehirns und der Hirnhaüte im Kindesalter, in Bruns, Cramer and Ziehen: Handbuch die Nervenkrankheiten im Kindesalter, Berlin, 1912, vol. 13, p. 678.

6. Trevelyan, E. F.: Some Observations on Tuberculosis of the Nervous System, Lancet **2**:1276, 1903.

With the former tabulations of cases from the general literature and with the estimates from postmortem examinations, it is interesting to compare the figures from individual clinics in which large numbers of brain tumors have been subjected to operation. Thus Tooth,[7] in 1912 and 1913, reported a series of 500 intracranial tumors from the records of the National Hospital, Queen Square, among which were fourteen tuberculomas (2.8 per cent). This small percentage was explained on the grounds that patients with tuberculomas were more likely to gravitate to children's and general hospitals, where, according to Tooth's expressed opinion, many of them may recover. Clarke,[8] in 1916, among ninety-nine cases of tumor in Dr. Spiller's collection in the University of Pennsylvania found six tuberculomas. Heuer and Dandy[9] in the same year gave a report of seventy tumors of which forty-two were verified, two of them being tuberculomas. Three years later, in a report from Eiselsberg's clinic covering the years from 1901 to 1919, Marburg and Ranzi[10] recorded the cases of 318 surgically treated patients with brain tumor, of which only a small percentage appear to have been verified. Of twenty cerebral tumors (excluding pituitary growths) there were two tuberculomas, and of twenty-two subtentorial tumors there were three others. Up to Oct. 15, 1925, approximately 1,000 cases of brain tumor had been verified in the Peter Bent Brigham Hospital clinic either by operation or by necropsy, with only fourteen tuberculomas (1.4 per cent) in the list. Patients of all ages are included, and the figures, therefore, are by no means restricted to an adult group, though adults predominate. For purposes of comparison with Starr's tables, the patients have been divided as he divided them, i. e., those under and those over 19 years of age. The results show that there have been 140 verified cases in the former group with five tuberculomas (3.5 per cent), and 860 cases in the latter group with nine tuberculomas (1 per cent). Starr's computation, as may be recalled, was 50.6 per cent and 13.6 per cent, respectively, for the two groups; and the present-day figures are in agreement with his estimates in 1895, in showing that tuberculomas still continue to be approximately four times more common in persons under 19 than in persons above that age.

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7. Tooth, H. H.: Some Observations on the Growth and Survival Period of Intracranial Tumors, Brain **35**:61, 1912; The Treatment of Tumors of the Brain, and the Indications for Operation, Tr. Seventeenth Internat. Cong. Med., London, 1913, sec. 7 (Surgery), part 1, pp. 203-299.

8. Clarke, F. B.: A Study of the Anatomic Location and Histopathology of Ninety-Nine Brain Tumors, Rev. Neurol. & Psychiat. **14**:485 (Nov.) 1916.

9. Heuer, G. J., and Dandy, W. E.: A Report of Seventy Cases of Brain Tumor, Bull. Johns Hopkins Hosp. **27**:224 (Aug.) 1916.

10. Marburg, O., and Ranzi, E.: Zur Klinik und Therapie der Hirntumoren mit besonderer Berücksichtigung der Endresultate, Arch. f. kin. Chir. **116**:96 (July 21) 1921.

The 140 examples of tumor in patients under 19 may be classified as shown in the accompanying table, which demonstrates that today, at least, the gliomas rather than the tuberculomas represent the 50 per cent of subtentorial tumors found in young people.

The infrequency of intracranial tuberculosis of any sort in adults compared with its occurrence in childhood often has been commented on, and naturally the incidence of tuberculomas is correspondingly small. The question naturally arises whether the greater prevalence of intracranial tuberculosis in young people and particularly of solitary tubercles may not have something to do with the type of organism concerned in the process, whether bovine or human; for it may be assumed that in some countries at least, as seems to be true in Scotland, the infection among children is usually milk-borne. So far as I am aware, no studies have been made to determine which of the two is more likely to produce the solitary tubercle type of lesion.

Classification of Tumors in Patients Under Nineteen

Cerebellar gliomas	73
Cerebral gliomas	26
Congenital tumors (suprasellar)	18
Chiasmal tumors	8
Pinealomas	5
Cerebellar tuberculomas	4
Pituitary adenomas	4
Pontile tuberculomas	1
Dural endotheliomas	1
	140

If the number of examples of a given disorder reported in the literature from decade to decade can be regarded as an index of the frequency of its occurrence, this phase of tuberculosis may be on the decline. Either interest in the subject has waned, or, what seems more probable in view of the drop in the incidence of tuberculosis in general, tuberculomas of the brain in this country at least have become comparatively rare.

TUBERCULOMAS IN THEIR SURGICAL ASPECT

Intracranial tuberculosis in any form has a notoriously high mortality. Surgical procedures have been largely unavailing either as a treatment of meningitis or for the removal of more or less circumscribed lesions. The reasons for this are readily apparent. In the first place, solitary tubercles are often multiple, Starr's estimate being 24 per cent, while Bruns [11] puts the figure at 60 per cent. In the second

11. Bruns, L.: Die Geschwülste des Nervensystems, ed. 2, Berlin, S. Karger, 1908.

place, tubercles even when single are often inaccessible to surgical removal, and decompressive operations are the only recourse. Nearly all writers on the subject have laid stress on the fact that the solitary tubercles of childhood show a marked predilection for the cerebellum and brain stem. Furthermore, a terminal meningitis, especially in children and therefore particularly in cerebellar cases, is frequent. Boltz [12] found meningitis in association with a solitary tubercle fifteen times in eighteen cases, and Trevelyan in twenty-three of thirty-three cases. Though the likelihood of meningitis apparently diminishes with increasing age, it is doubtless the terminal event, in the natural course of things, in almost all cases. If recoveries were common, healed tuberculomas would be encountered more often at necropsy, but this is exceedingly rare. Then, too, advanced tuberculosis in other parts of the body often precludes or seriously complicates surgical intervention, even though the intracranial lesion may be accessible and single.

A number of writers, impressed by the high mortality following surgical intervention, have compiled the cases in the literature in which the tuberculous tumor has been partly or wholly removed. A reperusal of the literature shows that many cases had been overlooked in preparing the tables, and it is hardly necessary now to go to the labor of correcting them or of bringing them up to date, particularly since the early operations they chiefly record were done almost exclusively for symptoms pointing toward a lesion of the paracentral convolutions of the cerebrum. For example, Allesandri,[13] in 1905, combining the figures of Chipault, Preyer, Duret and others, brought the number of surgically treated patients up to twenty-eight, only six of them presenting cerebellar lesions.

REPORT OF CASES

The surgical experiences with intracranial tuberculomas or presumed tuberculomas at the Peter Bent Brigham Hospital are comprised in the following seventeen case reports, which will be summarized briefly. They are divided into three groups: (1) those in which a lesion has been exposed and extirpation accomplished or attempted (six cases); (2) those in which the lesion either was not exposed or was not removed when exposed and its nature not positively verified till necropsy (eight cases); (3) examples of presumed tuberculoma not actually verified (three cases).

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12. Boltz, R. O.: Ein Beitrag zur Statistik und Anatomie der Tuberkulose im Kindesalter, Inaug.-Dis., Kiel, 1890, p. 21.

13. Allesandri, R.: Interventions chirurgicales dans la tuberculose de méninges et de l'encéphale, Cong. internat. de tuberc., Rap., Paris, 1905, pp. 201-222.

GROUP I (SIX CASES)

In group 1, as will be observed, an apparently successful extirpation of the lesion led in all cases to a prompt and immediate great improvement in the patient's condition, to be followed in five instances by tuberculous meningitis and death at intervals varying from four to ten weeks after operation. The five operations were all for cerebellar lesions. The sixth patient, in whom a cerebral lesion was removed, had a survival period of fifteen months with no evidence of local recurrence. The six cases are presented in the chronological order of the hospital admissions.

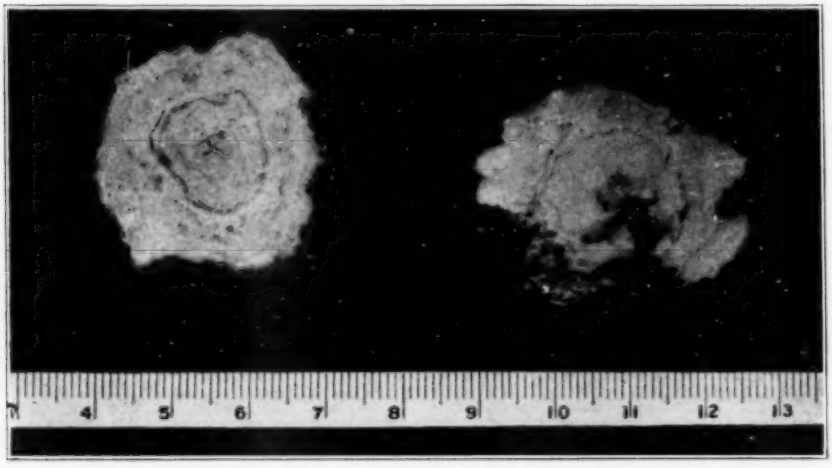

Fig. 1 (case 1).—Median section of two tuberculomas, showing the one on the right broken in removal.

CASE 1.—*Extirpation of two cerebellar tuberculomas from a child. Excellent operative recovery. Death ten weeks later. Necropsy showing tuberculoma of cerebral peduncle with widespread tuberculosis (bovine?) elsewhere.*

History.—The patient, E. M. F., aged 2 years and 11 months, was admitted on May 30, 1913, with a complaint of headaches and weakness of the left side of the body of about two months' duration. There had been no vomiting, but vision was thought to be impaired. The family history was unimportant. At the age of 13 months, the child had a prolonged diarrhea. Three months later the abdomen became tender and distended, and a diagnosis of tuberculous peritonitis was made. The symptoms subsided, but the child remained languid, lost weight, and at 23 months had a severe attack of measles with ensuing bronchitis. Since then there had been continued fever with recurrent attacks of diarrhea. Two months before admission, intracranial complications became apparent, at first taken to be tuberculous meningitis. The head became enlarged, the fontanel, which had never closed, became full, and there was marked twitching of the left side of the face and tremor of the right arm.

Fig. 2 (case 1).—Perfectly healed suboccipital wound.

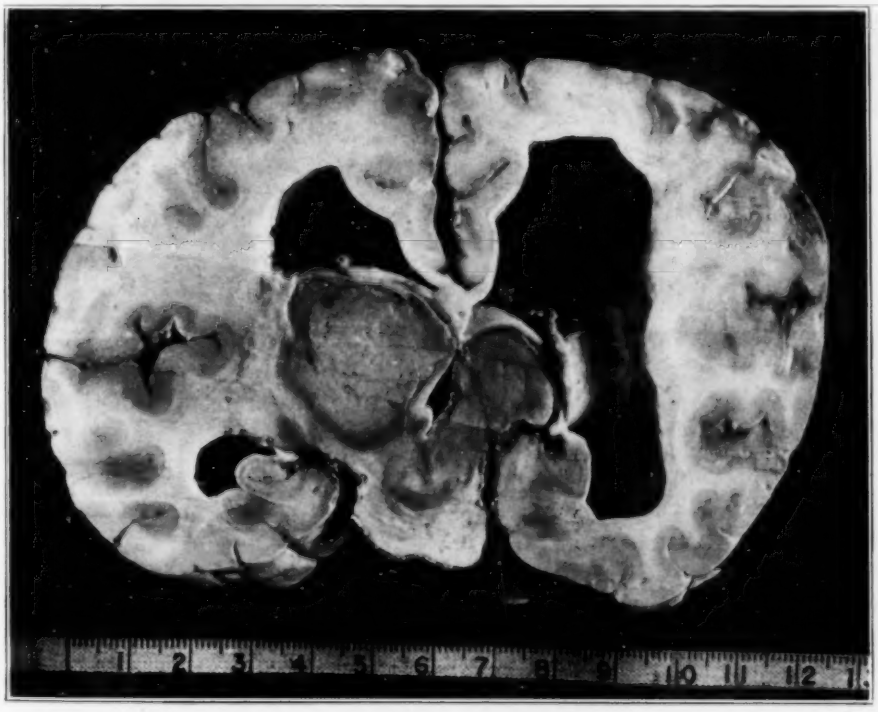

Fig. 3 (case 1).—Large tuberculoma of the left cerebral peduncle.

Examination on Admission.—The child was fretful and emaciated, with a left facial palsy, a low grade of choked disk and a large head evidently associated with separation of the cranial sutures. There was marked suboccipital bulging and tenderness. Slight exophthalmos was present, with divergent strabismus; no nystagmus was seen. The left side was hemiparetic, and the deep reflexes were exaggerated, with a positive Babinski sign. There was definite cerebellar incoordination with hypotonia. The child was unable to stand. Speech was thick and the deglutition reflexes were impaired. A left cerebellopontile tuberculoma was suspected.

Operation.—On June 3, a suboccipital exploration by Dr. Cushing revealed two large solitary tubercles situated in the left cerebellar hemisphere. The deeper lesion, which was adherent to the dura of the left cerebellar fossa and the tentorium, was broken in process of removal (fig. 1).

Course.—There was perfect healing of the wound (fig. 2), and the child did exceedingly well for six weeks, with marked amelioration of symptoms. The steady improvement was interrupted by a convulsion on July 18, and she began to lose ground. Incessant twitching of the muscles of the face, arm and leg on the right, with occasional convulsions and unconsciousness came on. The child died, August 15, seventy-three days after the operation..

Necropsy.—Examination showed active tuberculosis involving the bronchial lymph nodes, lungs, liver, intestines, uterus and ovary. There was a tuberculous meningitis, and the cerebral hemispheres contained multiple small tuberculomas. The cerebellar tissues at the site of the original extirpation apparently had healed completely, with no evidence of re-formation of the local tubercle. There was a large solitary tubercle (fig. 3) of the left cerebral peduncle.

Histologic studies of the organs, by W. T. Councilman, showed "tubercles forming in extremely resistant tissues; slow extension of the caseous necrosis with rapid cicatrization—conditions associated with strains of tuberculosis to which man is resistant, such as the bovine bacillus."

Comment.—A case of this sort has encouraging as well as discouraging features. The complete healing at the site of extirpation of the two large tumor masses and the absence of any other involvement of the cerebellum shows what might be accomplished under more favorable circumstances. On the other hand, the multiplicity of the lesions, the more serious one of which was located at the crossroads of the nervous system, shows the hopelessness of the malady in general.

Tubercles of the pontile protuberance are known to be common, and possibly the lesion there should have been clinically suspected in this case, though it was masked and its symptoms were overlaid by the evidences of cerebellar involvement. Many cases have been reported of tuberculomas involving the protuberance, causing what is known to the French as the "syndrome of Raymond and Cestan" accompanied by facial spasm.[14]

14. Aldin-Delteil and Levi-Velensi: Tubercle de la protubérance, Rev. neurol. **2**:464 (Oct.) 1925.

CASE 2.—*Extirpation of cerebellar tuberculoma from a young adult. Good recovery from operation. Death from meningitis five weeks later. Necropsy.*

History.—The patient, S. M., a Polish laborer, aged 22, admitted to the hospital, Jan. 8, 1915, two years previously had had a left-sided pleurisy requiring aspiration; since then he had had a persistent cough and night sweats. There was a brief history of right frontal headaches for six months, with vomiting at irregular intervals. More acute symptoms had been present for from three to four weeks. These consisted of stiffness of the neck, staggering gait, vertigo and ataxia of the right arm. There had been a loss of 25 pounds (11.3 Kg.) in weight.

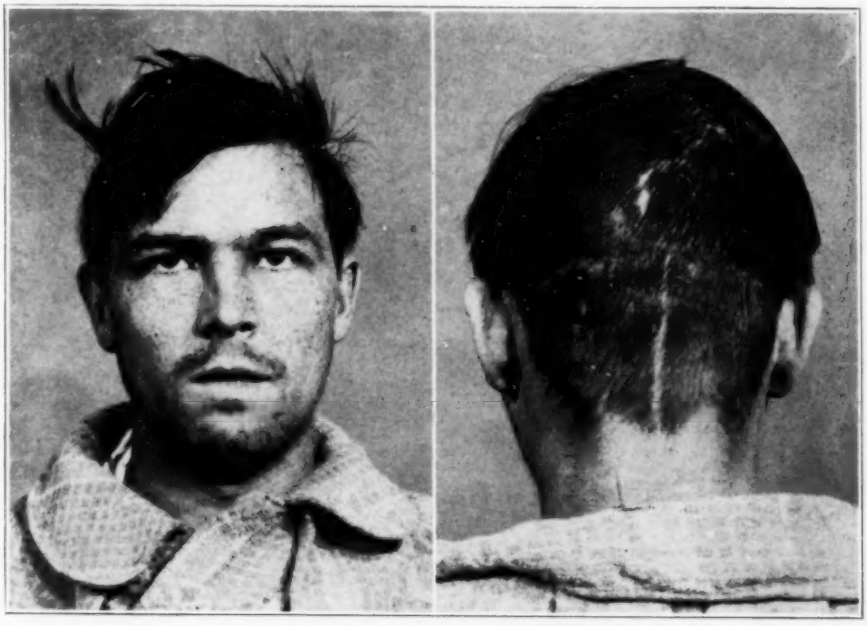

Fig. 4 (case 2).—Well healed wound three weeks after operation.

Physical Examination.—The man was emaciated, with signs of pulmonary tuberculosis. He had bilateral choked disk of from 4 to 5 diopters, and ataxic gait with a tendency to fall to the right. The right labyrinth was irresponsive to stimulation, and the head was tilted to the right shoulder. A subtentorial tumor (tuberculoma or glioma) was evident. The condition was so critical as to make operation imperative.

Operation.—On January 11, a cerebellar exploration by Dr. Cushing disclosed a firm palpable tumor mass deep in the right hemisphere. The lesion, which proved to be a solitary tubercle about 4 cm. in diameter weighing 20 Gm., was extirpated after transcortical incision. The mass was broken on removal, because it was attached to the tentorium, and a fragment was left behind.. The cerebrospinal spaces were opened during the procedure, and there was escape of fluid. A section from the surface of the lesion showed a typical tuberculoma with an enveloping zone of cerebellar tissue undergoing gliosis.

Course.—The patient made a good recovery from the operation; the wound healed perfectly (fig. 4) and the choked disks subsided. However, he gradually developed signs of meningitis and died February 16, about five weeks after operation.

Necropsy.—Postmortem examination showed advanced pulmonary tuberculosis with cavitation. The brain, removed after fixation with intracarotid injection, showed granular ependymitis with dilatation of the ventricles, but there was no macroscopic evidence of meningitis in either the cerebral or the spinal meninges. At the site of the operation, there was considerable softening and necrosis with a muscle implant on the raw cerebellar surface. No tubercles were found elsewhere in the brain.

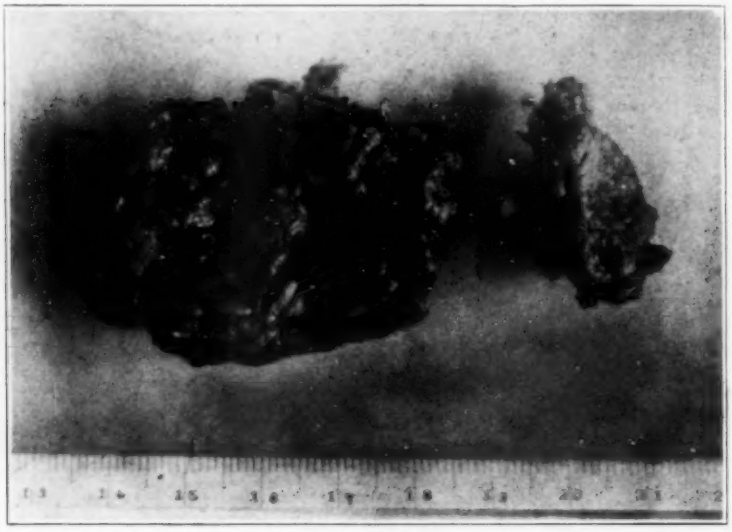

Fig. 5 (case 3).—Large tuberculoma removed in two pieces.

Comment.—This man was a poor risk, and if the diagnosis of tuberculoma had been certain rather than merely presumptive the operation in all probability would not have been undertaken. Even so, it shows how well these major procedures are tolerated by a patient in an advanced stage of pulmonary consumption; had the growth proved to be of another type, he might have been given a chance to fight off his constitutional disease.

CASE 3.—*Extirpation of cerebellar tuberculoma from a child. Excellent temporary recovery. Death from meningitis six weeks later.*

History.—The patient, H. B. M., a girl, aged 7, was admitted to the hospital, May 7, 1915, with a family history of tuberculosis. She had had measles and mumps in early childhood; also a severe attack of whooping cough with high fever one year prior to entrance, with confinement in bed for five or six weeks. The weight dropped from 64 to 37 pounds (29 to 16.8 Kg.). The chief complaint was fronto-occipital headaches for five months, with stiffness of the neck

Fig. 6 (case 3).—Child three weeks after operation, after treatment under heliotherapy.

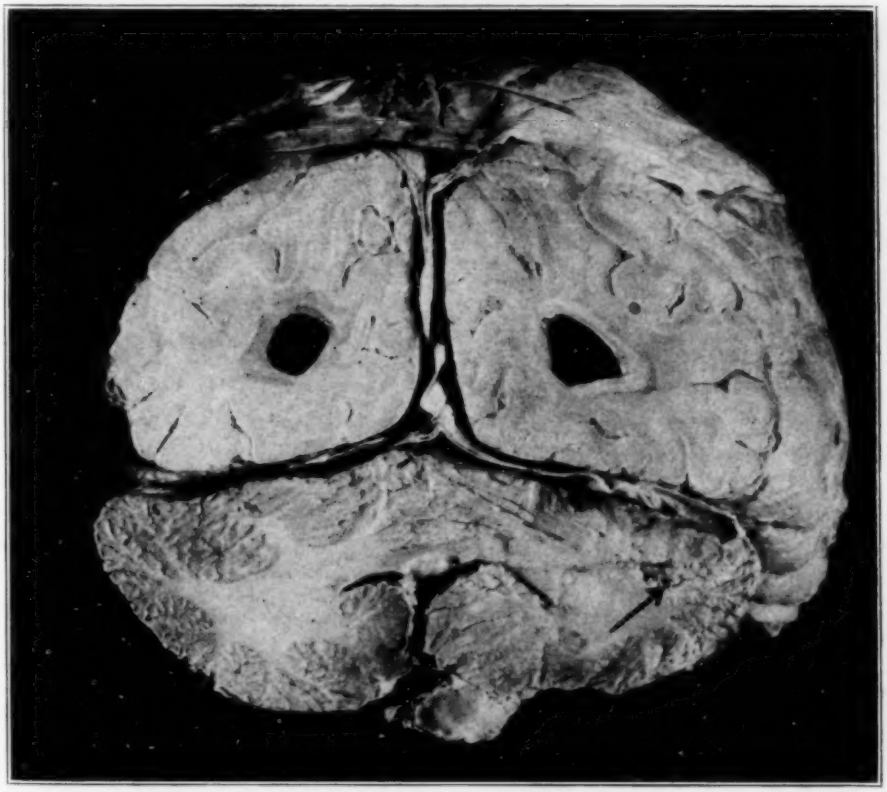

Fig. 7 (case 3).—Site from which large tuberculoma had been removed six weeks previously.

and left suboccipital tenderness. During this time there had been diplopia with internal squint on the right. Vision had been failing for three weeks, particularly in the left eye, and vertigo with staggering to the right had been observed.

Physical Examination.—This revealed bilateral choked disk of from 4 to 5 diopters, with secondary atrophy more marked on the left; the right pupil was larger than the left, with loss of reaction to light and in accommodation. There was a sustained nystagmus, coarser on looking to the left, a right sixth nerve paralysis, slight ataxia of both hands and positive Romberg sign with swaying to the right. The skull gave a "cracked pot" sound on percussion, indicating separation of the sutures. A tuberculin test (Pirquet) was positive. The diagnosis was tuberculoma of the cerebellum.

Operation.—On June 4, Dr. Cushing exposed a large intracerebellar tuberculoma in the right hemisphere by a transcortical incision and extirpated it. Unfortunately, it was broken in two pieces which were removed separately (fig. 5).

Course.—The early postoperative course was excellent in all respects. The child was up and about, and was kept in the sun until thoroughly pigmented (fig. 6). The temperature remained normal until about July 1, when she began having an evening rise in temperature and some stiffness of the neck. From this time on there was a downward trend in the condition, and soon meningeal symptoms became evident. Lumbar puncture disclosed fluid under tension, with 152 cells, chiefly polymorphonuclear. Guinea-pig inoculation was positive for tuberculosis. She died on July 19, forty-five days after the operation.

Necropsy.—This showed chronic tuberculosis of the mesenteric lymph nodes, with evidences of old quiescent peritoneal involvement. The leptomeninges were edematous and showed miliary tubercles. There was marked internal hydrocephalus with granular ependymitis.

On section of the brain, an area was disclosed beneath the right ventricle resembling an infiltrating glioma in its appearance, subsequently shown to be edema with perivascular hemorrhage. Sections through the cerebellum (fig. 7) showed that the operative field was largely healed, despite an unmistakable small area which still contained a fragment of the original tuberculoma broken off in process of removal. Most of the organs, the liver, spleen and lungs, showed miliary tuberculosis.

Comment.—For this child there would have been no possibility of recovery without operation. Whether decompression alone would have improved her chances in view of the large size of the lesion is doubtful. Complete removal of the growth, together with the adherent and involved meninges in view of its deep-seated and inaccessible position, was out of the question.

CASE 4.—*Extirpation of large cerebellar tuberculoma from a child. Favorable surgical recovery. Death from meningitis one month later.*

History.—The patient, J. G., a child, aged 4, was admitted to the hospital, Feb. 20, 1917, because of rapid loss of vision and staggering gait. The family and past history were unimportant. About two months before entrance, failing vision with dizziness and impairment of gait were first noticed.

Physical Examination.—The child was well nourished, cooperative and without subjective complaints. There was no history of headache or vomiting at any time. There were complete blindness from secondary optic atrophy, separation

of the sutures of the skull, sustained nystagmus with the quick component to the right, exaggerated knee reflexes with bilateral clonus and Babinski sign, positive Romberg sign and excessive instability of cerebellar type. The diagnosis was cerebellar tumor.

Operation.—On February 27, Dr. Cushing made a suboccipital exploration disclosing hydrocephalus and great tension. When the dura was incised, a hard, edematous area occupying most of the right cerebellar hemisphere was disclosed. Because of respiratory difficulties it was necessary to remove the arch of the atlas in order to liberate the medulla. The large tumor mass was then freed and enucleated

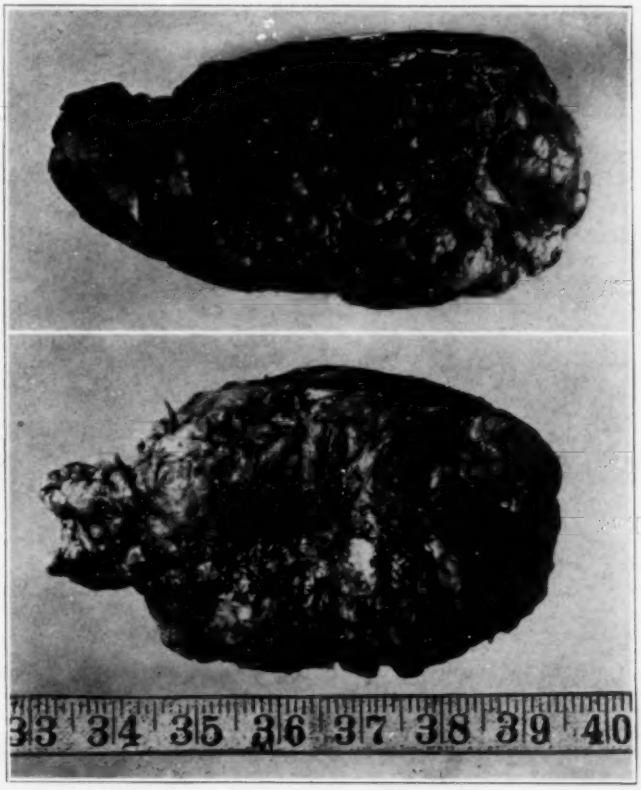

Fig. 8 (case 4).—Large tuberculoma (natural size) removed from the cerebellum of a child of 4 years.

(fig. 8). The raw cavity was superficially fixed with Zenker's solution. The removal of the tumor laid bare the tentorium, on the surface of which there were definite tubercles showing that an adhesive local meningitis was already under way. The ventricular fluid injected into a guinea-pig was negative for tuberculosis.

Course.—The child made an excellent early postoperative recovery; the wound healed perfectly; she was given sun baths and was kept outdoors. On March 13, she began to have a slight rise in the afternoon temperature; she became somewhat irritable, showed a positive Kernig sign, became comatose, and died, March 28, thirty days after operation. Necropsy was refused.

Comment.—In this case tuberculoma was not suspected until the lesion was exposed at operation. Just what should have been done with it under the circumstances is one of the matters under consideration in this paper.

CASE 5.—*Extirpation of a corticomeningeal tuberculoma from the cerebellum of a young adult. Excellent operative recovery; discharge from hospital; reentry and death from meningitis eight weeks after operation. Necropsy.*

History.—The patient, S. D., a man, aged 27, was admitted to the hospital, July 30, 1919. He had had measles, whooping cough and mumps in childhood, severe typhoid with relapse when 13 years of age, and in 1916 had contracted syphilis. A year before entrance, while he was in the army, the left testis had been removed. The present illness began four months before admission with stiffness of the neck. Soon there were severe pains in the suboccipital region

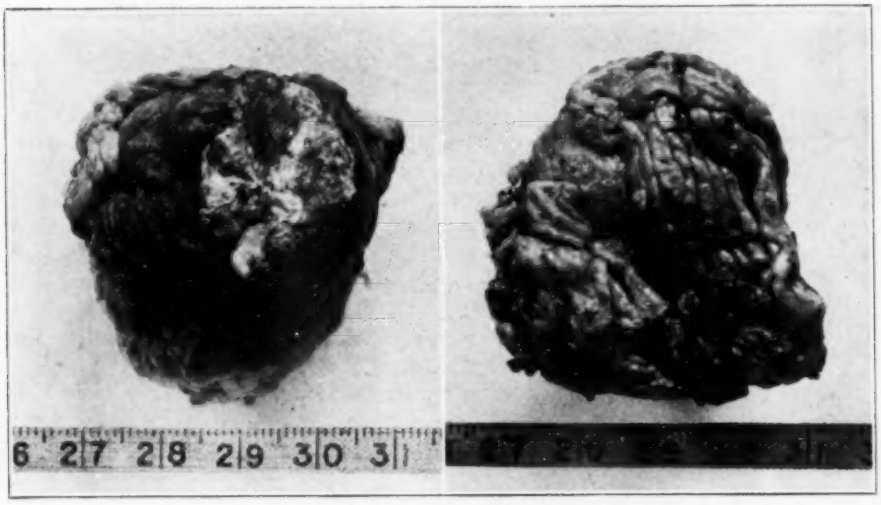

Fig. 9 (case 5).—Tumor as removed (natural size) fully enveloped in cerebellar tissue and with adherent patch of dura shown in figure to left.

accompanied by vertigo and vomiting. Some unsteadiness of gait ensued with a tendency to stagger to the right. He had lost 13 pounds (5.9 Kg.) in three months.

Physical Examination.—The man was bedridden, and in terror of pain in the suboccipital region which was brought on by the slightest change of position. Movement of the head caused uncontrollable cervical retraction with slowing of pulse and respiration. There was bilateral choked disk of 1 diopter, slight occasional nystagmus, a slowed pulse, ataxia of the arms and a positive Babinski sign on the left. The Wassermann reaction was negative in the blood. In the two days he was under observation, a slight left facial weakness, with increasing disability of the left side more than the right, developed. Gait and station were impossible to test. The diagnosis was left cerebellar tumor.

Operation.—On August 1, an emergency suboccipital exploration under procaine hydrochloride was performed by Dr. Cushing, and a firm, left-sided tumor, 4 cm. in diameter, adherent to the dura, was carefully enucleated in its totality, enveloped

in a shell of attached cerebellar tissue (fig. 9). On section it proved to be a tuberculoma (fig. 10).

Course.—There was perfect healing of the wound, and the postoperative course was most promising for the ensuing three weeks, with complete subsidence of the choked disk. It was observed, however, as soon as the patient began to move about that exertion would cause low fever. He was finally discharged September 12, six weeks after the operation, plans having been made for his transfer to Saranac Lake. On the way to the train he felt ill and returned to the hospital. From this time he began to fail. An incision was finally made through the muscles down to the site of the tumor extirpation; some turbid fluid was secured, which proved positive for tubercle bacilli. Drainage was instituted without avail, and death occurred September 24, nearly eight weeks after the operation.

Necropsy.—This disclosed an active tuberculosis of the apex of the right lung, with a healed process at the left apex; tuberculous bronchopneumonia, and extensive genito-urinary tuberculosis involving the prostate and seminal vesicles. The brain showed a basilar tuberculous meningitis. There were no other solitary tubercles.

Fig. 10 (case 5).—Section of tuberculoma removed intact from cerebellum (natural size).

Comment.—This patient was in a critical condition, and the localizing diagnosis was made largely on the subjective symptoms rather than on any objective cerebellar signs. The operation was undertaken as an emergency procedure on the second day after admission before the laboratory tests could be completed, and a probable diagnosis lay between gumma and tubercle. It was subsequently learned that the testis had been removed for tuberculosis. Dr. Cushing's operative note states that when the firm lesion adherent to the dura was first brought to view, it was mistaken for a meningioma, and its actual pathologic condition was not assured until the tumor was sectioned after removal.

That for the first three weeks the patient should have been up and about with a normal temperature and free from his former distressing symptoms is remarkable. When he was discharged on the forty-second

day, he was thought to have passed safely the critical period of meningeal infection. The exertion incidental to his departure from the hospital was evidently the turning point in the case.

CASE 6.—*Extirpation of a solitary tubercle from the cerebrum of a middle-aged woman. Excellent surgical recovery. Death from peritoneal tuberculosis fifteen months later. No necropsy.*

History.—The patient, A. C., aged 48, the mother of six children, was admitted to the hospital, Jan. 29, 1923, complaining of convulsive seizures and weakness of the right side. Seven years previously she had begun to have repeated attacks of cervical adenitis. Some of the inflamed glands were incised; others opened spontaneously. These continued to discharge until two years before admission. For nine months she had had a reddish area on the right cheek which had discharged pus, evidently a tuberculous dermatitis. She had otherwise enjoyed perfect health until the onset of the present malady. For more than two years she had had mild jacksonian seizures beginning in the right hand and spreading to the arm and leg, associated with a progressing spastic palsy. During the six months prior to entry, there had been seven convulsions with unconsciousness. Objectively there was noted a marked "lead-pipe" spasticity of the right arm and leg with restricted movement of all muscle groups, associated with exaggerated deep reflexes and a positive Babinski sign. Complete astereognosis was present, with loss of postural sense. There was no fever and no choked disk. Mentality was somewhat dulled, but there was no aphasia. The diagnosis was left superior parietal tumor (meningioma?).

Operation.—On February 6, Dr. Cushing exposed a left parasagittal tuberculoma, the size of a hen's egg, adherent to the meninges. This was removed by tilting it out much as one would deal with a parasagittal meningioma. The dura, which was slightly adherent to the growth, when stripped away was found dotted with many fresh tubercles. On section, the tumor (fig. 11) showed a central area of necrosis surrounded by a translucent, gray, evidently fresh spreading process.

Course.—It was expected that this procedure would lead to a rapid meningeal infection. On the contrary, the patient made an excellent postoperative recovery in spite of a temporary increase in the contralateral symptoms, together with slight paraphasia. From this she had practically recovered by the time of her discharge, March 7, four weeks after the operation. Daily Alpine lamp treatments had been given over the body surfaces, and she was advised to continue this after returning to her home.

She did extremely well for the next year. A report of March 31, 1924, states that the otherwise excellent physical condition up to this time had recently been somewhat affected by certain curious spasmodic discomforts involving the calf muscles and toes on the left side, diagnosed as sciatica. Soon abdominal symptoms supervened, with distention and obstruction. Laparotomy was performed on May 6, disclosing peritoneal tuberculosis. She died the following day, fifteen months after the extirpation of the cranial tuberculoma.

Comment.—Although this woman had obvious tuberculous adenitis and a patch of lupus on her face, physical examination showed no signs of tuberculosis elsewhere in the body, and the fact that the jacksonian seizures had existed for more than two years caused the possibility of a tuberculoma to be discounted in favor of a benign tumor of the endothelial type.

Fig. 11 (case 6).—Tuberculoma on section adherent to dura in parasagittal region.

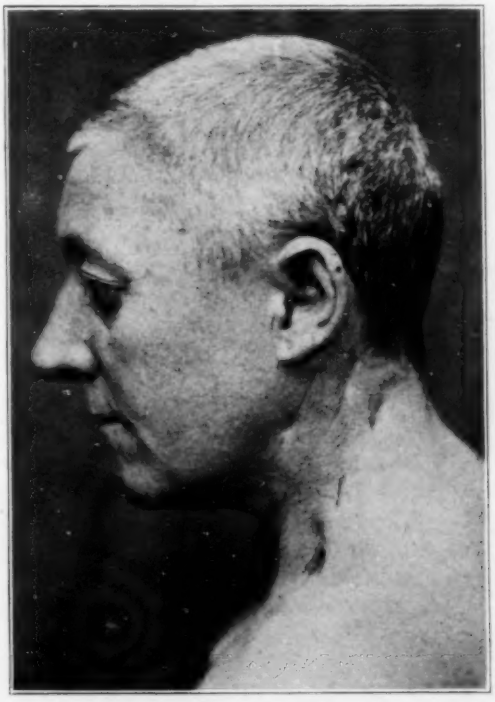

Fig. 12 (case 6).—Patient three weeks after removal of parasagittal meningioma. Scars of tubercular adenitis should be noted. The site of the osteoplastic resection is inconspicuous.

It is to be noted that this was one of the few examples of tuberculoma of the cerebrum in the series, and the only case, furthermore, which may be looked on as a possible cure of the local lesion by removal. The patient at least had no further intracranial symptoms, although she ultimately succumbed to tuberculous peritonitis more than a year after the cerebral granuloma was extirpated.

GROUP II (EIGHT CASES)

There follow the additional histories of eight cases in which the lesion was either not disclosed until necropsy or was left in situ without interference on being recognized as a tuberculoma, with the hope that the decompressive measure might alleviate symptoms. The eight cases are given in the chronologic order of their hospital admission.

CASE 7.—*Cerebral exploration, for presumed tumor, in a child. Hydrocephalus disclosed, and callosal puncture made. Death after five weeks. Necropsy, disclosing midcerebellar tuberculoma.*

History.—The patient, D. B. R., a boy, aged 7, was admitted to the hospital, March 26, 1913, with the history of a bad fall, two years before admission, in which he struck the head, but with no subsequent symptoms. In July, 1912, he had what was called a "heat stroke" followed by a series of four convulsive attacks. Headache and backache with vomiting and diplopia had gradually appeared in the interval.

Physical Examination.—The child was irritable, with choked disks of 4 diopters and convergent squint. Limitation of upward movement of the eyes was noted by one observer. There was no ataxia or instability, and the Romberg sign was negative. There was no nystagmus. The skull had a "cracked-pot" sound, and there was a bruit appreciated by the patient and heard by the stethoscope, synchronous with the pulse, over the parietal region. The diagnosis was unlocalized cerebral tumor.

Operation.—April 3, an exploratory craniotomy was performed by Dr. Bagley, disclosing marked hydrocephalus. Callosal puncture was performed for palliative purposes.

Course.—No improvement followed this procedure, and on April 23, increased backache with a positive Kernig sign suggesting meningitis became apparent. A series of lumbar punctures was performed, large amounts of fluid being secured under pressure, with a high cell count, up to 375 cells on one occasion. No tubercle bacilli were found, and guinea-pig inoculation proved negative.

The child was finally discharged against advice with the diagnosis of pseudotumor. He died, May 12, thirty-nine days after the operation.

Necropsy.—The brain was forwarded for study. It showed a solitary tubercle, 2.4 cm. in diameter situated in the cerebellar vermis. The meninges contained miliary tubercles with an abundance of acid-fast bacilli.

Comment.—What was found at necropsy in this case was entirely unsuspected. There had been nothing to suggest definitely the presence of a subtentorial tumor, although before the child's discharge a tuberculous meningitis had been suspected.

CASE 8.—*A conglomerate tuberculous mass involving the left cerebral hemisphere of a young adult, exposed at operation. Death ten days later. Necropsy: multiple tuberculomas.*

History.—The patient, William P., aged 21, was admitted to the hospital, Feb. 13, 1914, with a definite family history of tuberculosis. In spite of an attack of adenitis as a child and a long-standing arthritis of the right shoulder, his health had been considered good until six weeks before admission, when he began to have numbness and weakness of the right hand and arm, with slowness of speech and difficulty in swallowing—symptoms that had advanced rapidly. There had also been some cough, slight fever, and loss of weight, and recently severe headaches, nausea and vomiting.

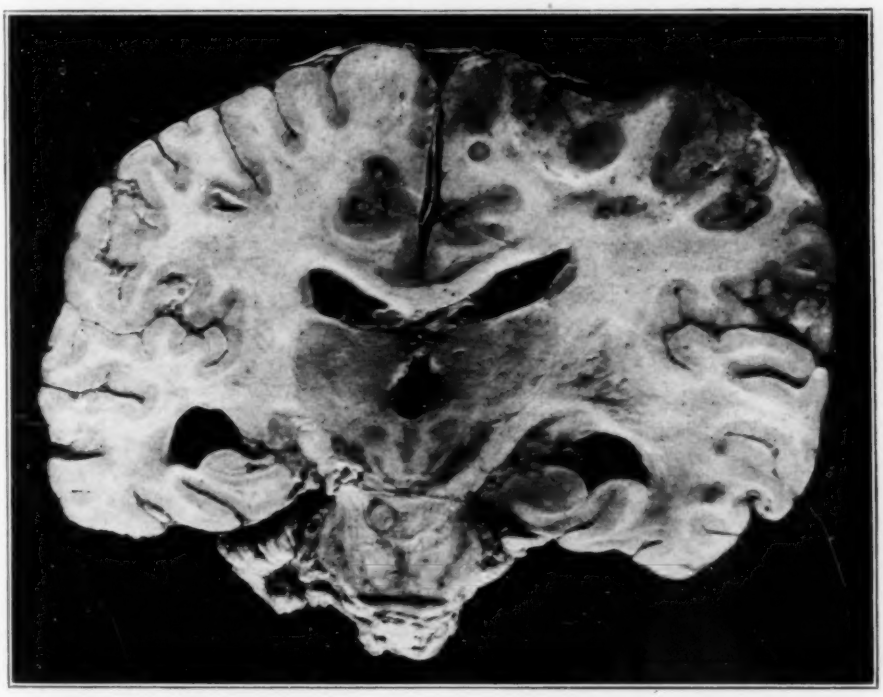

Fig. 13 (case 8).—Multiple small tuberculomas and a conglomerate mass in the left parietal cortex.

Physical Examination.—The man was undernourished, with evident pulmonary tuberculosis and a right-sided flaccid hemiparesis with paresthesia, astereognosis and increased reflexes. There were bilateral choked disk of from 4 to 5 diopters; bilateral exophthalmos; nystagmus, and normal visual fields. The diagnosis was left cerebral tuberculoma.

Operation.—As the patient was suffering greatly, Dr. Cushing made a left osteoplastic exploration, which disclosed a granulomatous mass adherent to the under surface of the dura. No attempt was made to do more than give the tense brain a generous decompression.

Course.—Death occurred ten days later.

Necropsy.—This showed old tuberculous lesions of the lungs, bronchial glands and epididymis, with an acute miliary process involving most of the organs of the body. The brain showed a most extensive process with a multitude of small tuberculomas (fig. 13) in the cerebrum, pons and cerebellum. The lesions were undergoing caseation, and there was extensive cerebral edema with patches of necrosis.

Comment.—From a surgical standpoint, operation in such a case could have been justified only for purposes of euthanasia; if the extent of the intracranial process could have been anticipated, the operation would not have been undertaken.

CASE 9.—*Suboccipital decompression for cerebellar tumor in an adult, with early fatality. Necropsy: pontile tuberculoma.*

History.—The patient, F. O. K., a Russian Jew, aged 30, admitted to the hospital, June 16, 1914, was said to have begun to have headaches four months before admission. Then he developed a cough and consulted a physician, who found a right-sided pleural effusion, which was tapped. He had had considerable tenderness, soreness and pain in the suboccipital region associated with blurred vision, diplopia, dizziness and unsteadiness in gait.

Physical Examination.—There were early choked disks and slight nystagmus. There was marked instability, a positive Romberg sign and considerable difficulty in deglutition. A roentgenogram of the chest showed clouding of the apexes of both lungs.

During the next ten days the subjective symptoms became accentuated, together with an increase of ataxia, nystagmus and other symptoms pointing chiefly to the cerebellum, though a definite limitation of vertical movements of the eyes was noted by several observers. Throughout the hospital sojourn, the patient had a normal temperature. The diagnosis was cerebellar (or pontile?) glioma.

Operation.—On July 1, 1914, a cerebellar exploration was performed by Dr. Cushing. Nothing of importance, with the exception of tension, was found.

Course.—The patient became cyanotic, had Cheyne-Stokes respiration and elevation of temperature to 104 F. Death occurred from respiratory failure the following day.

Necropsy.—The postmortem examination disclosed generalized tuberculosis involving the lungs, pleurae, pericardium and kidneys. The brain showed no abnormalities except enlargement of the pons. It was not until a series of coronal sections were made that a large pontile tuberculoma (fig. 14) extending backward into the cerebellar vermis was disclosed. The meninges were free from involvement.

Comment.—This patient's pulmonary tuberculosis seemed quiescent, and an intracranial tubercle apparently was not suspected. The cause of death was doubtless the shift in the position of the lesion induced by the suboccipital decompression, a complication that is particularly likely to occur when there is a pontile lesion.

CASE 10.—*Subtemporal decompression for early choked disks in a middle-aged man. Necropsy three months later disclosed a cerebellar tuberculoma.*

History.—The patient, J. J. P., a laborer, aged 53, entered the hospital, April 20, 1914, complaining of headache, vomiting, vertigo and a tendency to stagger toward one side for about six weeks.

Physical Examination.—The patient had slightly hazy disk margins, anisocoria, and impaired hearing on the right, with some unsteadiness of gait and a slightly positive Romberg sign. A cerebellar tumor was suspected; but surgical intervention was postponed, and he was discharged. He returned two months later with

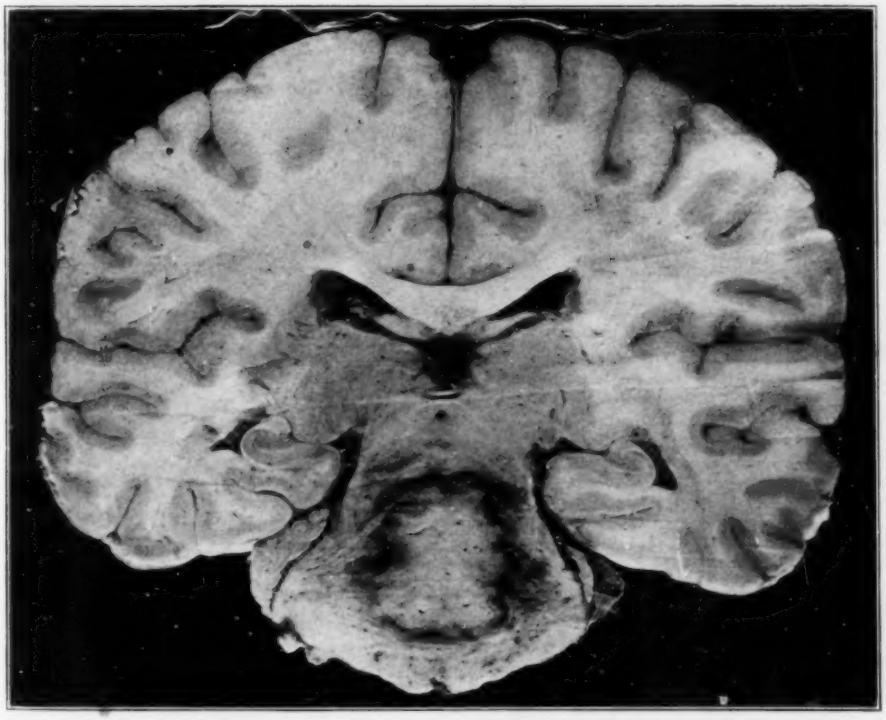

Fig. 14 (case 9).—Large central solitary tubercle of pons disclosed at necropsy, in a patient in whom a cerebellar glioma was suspected.

a continuance and aggravation of headaches, bilateral choked disks with hemorrhages, and marked mental changes.

Operation.—July 13, a simple right-sided subtemporal decompression was made by Dr. Cushing.

Course.—The choked disks subsided, and the headaches were relieved. He was discharged, July 26, and was not heard from subsequently. Death was reported on October 20.

Necropsy.—An unrestricted examination performed at the patient's home showed practically normal organs with no important abnormalities; there were no evidences of tuberculosis. A series of coronal sections of the brain showed slight hydrocephalus with a large granuloma in the vermis. Microscopic examination showed typical conglomerate tubercle with extensive caseation.

Comment.—This was a patient in middle age in whom a tuberculoma was not suspected, and the localization of the lesion in the cerebellum was not sufficiently definite to justify more than a subtemporal decompression to relieve pressure. He lived seven months after the outset of the early intracranial symptoms. The postmortem disclosures of a tuberculoma in the cerebellar vermis was unexpected, and the primary focus of tuberculosis was undetermined. The following case was essentially similar:

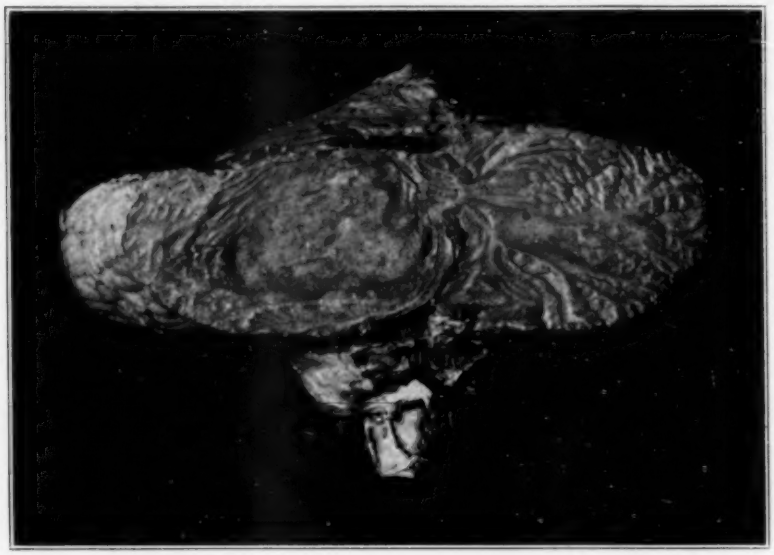

Fig. 15 (case 11).—Large unsuspected solitary tubercle of left cerebellar hemisphere disclosed at necropsy.

CASE 11.—*Right subtemporal decompression for unlocalized tumor in a middle-aged man. Death in three weeks with cerebellar tuberculoma found at necropsy.*

History.—The patient, George C., aged 45, was admitted to the hospital, June 15, 1914, with the history of having had pneumonia five months previously, followed by headaches and stiffness of the neck which had been severe enough to keep him confined subsequently. There had been failure of memory and, of late, obscuration of vision.

Physical Examination.—The patient was emaciated, bedridden and disoriented, and was suffering from extreme headache and bilateral choked disk of from 6 to 7 diopters. The gait and station could not be tested, but there was no demonstrable incoordination or nystagmus, and no suspicion of a cerebellar lesion was aroused. The deep reflexes were exaggerated throughout. There was no fever; thoracic examination was negative except for slight dulness over the left side of the chest; the temperature was normal. No suspicion of tuberculosis was aroused. The diagnosis was unlocalized intracranial tumor.

Operations.—On June 20, a subtemporal decompression was performed by Dr. Cushing. This gave only temporary relief, and on the assumption that there might be a frontal tumor an osteoplastic exploration was made on July 2. Nothing of importance was found except slight hydrocephalus.

Course.—No benefit was derived from the operations, and the man died from respiratory failure on July 14.

Necropsy—Postmortem examination, restricted to the brain, showed that the left hemisphere of the cerebellum was larger and harder than the right, with a marked pressure cone. Coronal sections disclosed internal hydrocephalus and a solitary tubercle (fig. 15) 3 cm. in diameter, with central caseation. There was no meningitis.

Comment.—This patient presumably had an unrecognized tuberculosis. He was too ill to subject to a satisfactory neurologic examination, and the situation of the lesion was not recognized. This case is an example of a large solitary tubercle of the cerebellum which had caused death, in the absence of meningitis, from pressure symptoms following two misdirected operations.

CASE 12.—*Suboccipital exploration with decompression for a presumed cerebellar tuberculoma in an adult. Death three months later. Necropsy: solitary tubercle involving vermis.*

History.—The patient, M. L. T., a married woman, aged 34, was admitted to the hospital, May 27, 1920. She had been operated on for tuberculous glands in the left side of the neck eight years previously. She had been married eleven years and had four healthy children, the last having been born the previous December. During the last pregnancy, she had an attack of influenza (?) followed by pleurisy with effusion requiring four aspirations. She had a normal parturition, but subsequently her general health began to fail. In January, 1920, she began having severe fronto-occipital headaches with nausea and vomiting. Vertigo soon was noted and a little later unsteadiness of gait with staggering to the left, stiffness of the neck, ataxia of the left arm and blurred vision with occasional diplopia. The outstanding complaint was of severe suboccipital pain.

Physical Examination.—The patient was ill-nourished, anemic and bedridden, with a puckered scar on the left side of the neck and obvious signs of pulmonary tuberculosis. There was bilateral choked disk of from 3 to 4 diopters, with secondary optic atrophy, unequal pupils, marked nystagmus and motor incoordination of cerebellar type chiefly marked on the left, with past-pointing and excessive instability. There was extreme suboccipital tenderness with cervical rigidity and slight paresis of the right side of the face. The probable diagnosis was cerebellar tumor, almost certainly tuberculoma.

Operation.—To relieve the extreme suffering, a suboccipital exploration was performed under procaine hydrochloride by Dr. Cushing, June 7. This revealed internal hydrocephalus with normal fluid, negative for tubercle bacilli (guinea-pig). The left cerebellar hemisphere was greatly enlarged, and an exploratory puncture disclosed a resistant tumor in the depths.

Course.—The operation afforded immediate great relief, with cessation of headache, vomiting, and subsidence of the choked disk. The patient was able to be up (fig. 16) for the first time in many weeks and was discharged, June 28, greatly improved. She died at home in Springfield two months later.

Necropsy.—A postmortem examination was limited to the head. The brain was removed and forwarded to us for study. It revealed a large tuberculoma which had not undergone caseation and which involved chiefly the cerebellar vermis (fig. 17). In addition there was a terminal tuberculous meningitis.

Comment.—This is a good example of the palliative effects of decompression alone in one of these cases. An attempt to remove this particular tumor, if its position had been more favorable, probably would not have changed the course of the disease.

Fig. 16 (case 12).—Patient on discharge, three weeks after cerebellar decompression. The absence of suboccipital protrusion should be noted.

CASE 13.—*Osteoplastic exploration and decompression in a child, for presumed tumor of the left hemisphere, with early fatality. Necropsy: solitary tubercle of left internal capsule.*

History.—The patient, S. L., an Italian boy, aged 8, was admitted to the hospital, Oct. 27, 1920, with a practically negative past history. The present illness had begun in June with a rather sudden onset of weakness of the right arm and leg, which had progressed to a partial hemiplegia. Severe frontal headaches with blurring of vision, vomiting and diplopia had occurred more recently.

Physical Examination.—The child was well nourished but excessively irritable, and complained bitterly of headache. There was bilateral choked disk of 4 diopters, with near-blindness, and an internal squint in the left eye with a partial ptosis and immobile pupil. There was some nystagmus. The lower part of the face, arm and leg on the right were hemiparetic, and the reflexes on this side were increased, with ankle clonus, a positive Babiński sign and other symptoms. The diagnosis was probable deep tumor of the left hemisphere (possibly pontile?) of an undetermined nature.

Operation.—A left cerebral exploration with subtemporal decompression was made by Dr. Cushing, October 30. Nothing of importance was found except hydrocephalus, the fluid showing 120 cells per cubic millimeter.

Course.—Respiratory difficulty with Cheyne-Stokes respiration led to death the following morning.

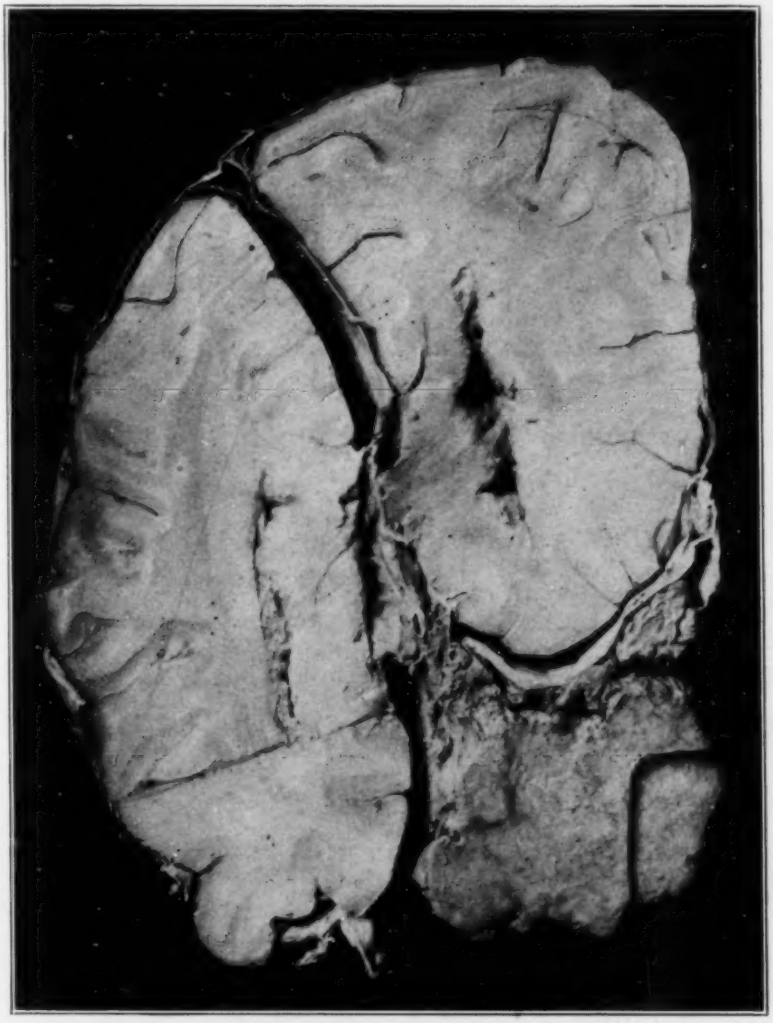

Fig. 17 (case 12).—Distorted brain due to removal without previous fixation. The section shows the large tuberculoma beneath the tentorium, most of the overlying cerebellar tissue having been stripped away.

Necropsy.—A postmortem examination was restricted to an examination of the brain. The external appearances were normal except for a marked pressure cone. A series of coronal sections showed a solitary tubercle measuring 2.8 by 3 cm. (fig. 18), which occupied the posterior part of the left thalamus and compressed

the internal capsule. Covered by a thin film of ependyma, the growth projected directly into the third ventricle which was pushed well off to the right. The lining of the ventricle was distinctly granular, showing that a tuberculous ependymitis was already under way. The tumor pressed down on and considerably flattened the pons. The iter was completely obstructed.

Comment.—In this child, tuberculosis was not suspected, and owing to the restricted necropsy the primary focus of the disease remains unde-

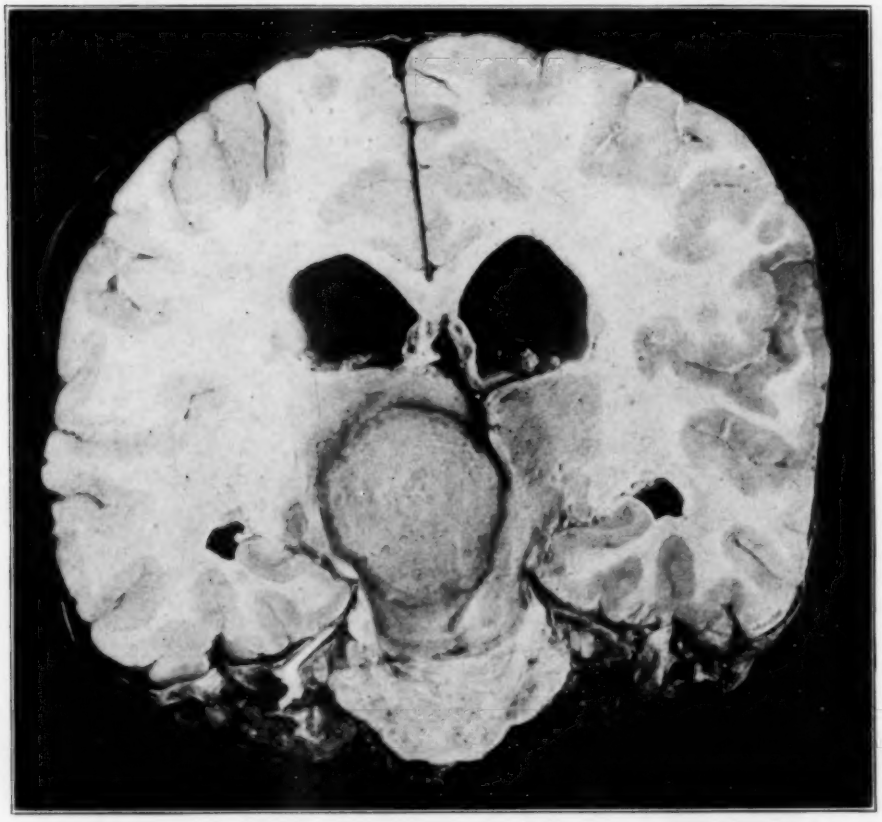

Fig. 18 (case 13).—Solitary tubercle chiefly occupying the left thalamocapsular region.

termined. The operation was undertaken in the expectation that there might be a glioma or gliomatous cyst deep in the left hemisphere, though there were some localizing features suggesting a subtentorial lesion. The clinical picture is now easily recognized as the so-called Weber syndrome; that is, a third nerve palsy with contralateral hemiparesis.

CASE 14.—*Suboccipital exploration for presumed tumor in a young woman, with fatality in seven weeks. Necropsy: tuberculoma of the restiform body projecting into the fourth ventricle with hydrocephalus.*

History.—The patient, L. E.,[15] a stenographer, aged 20, was admitted to the hospital, March 24, 1923, with the history of having been exposed to tuberculosis during the illness of her father, who had died shortly before. Otherwise, the past history had been unimportant except for frequent epistaxis and ten or twelve "fainting attacks" during the last five years. There had recently been a loss of 40 pounds (18.1 Kg.). The present illness began about twelve months prior to entry with influenza and an abscess in the lumbar region which ruptured spontaneously and healed. Attacks of vomiting, at times projectile, diplopia on occasions, headaches and blurred vision and dizziness and unsteadiness had been noted for three or four months. She showed a positive Pirquet skin reaction for tuberculosis.

Physical Examination.—The positive neurologic observations were listed on admission as follows: Bilateral papilledema of low degree, diplopia, photophobia with lacrimation, marked bilateral rotary nystagmus, weakness of the left external rectus, peripheral left facial weakness and weakness of the left hypoglossus. There was also a positive Romberg sign with staggering gait, and marked hypotonia particularly of the left arm, but without much incoordination. While the patient was under observation, an involvement of the left trigeminus was detected, and the reflexes on the left became exaggerated, with clonus and a positive Babinski sign.

Owing to headaches and incessant vomiting the condition was pitiable, and it was hoped that a suboccipital decompression might relieve them. The diagnosis was not assured, though a left cerebellopontile tuberculoma was naturally suspected because of the multiple crania nerve involvement of that side, the eighth, however, having escaped.

Operation.—On April 14, cerebellar exploration was reluctantly performed by Dr. Cushing, with nothing of importance found. There was no tension; the nerves in the lateral recess were normal in appearance. There was a grayish arachnoid with an excess of fluid which led to a provisional diagnosis of arachnoiditis.

Course.—Though the patient did well for a time (fig. 19) and remained free from headache, the postoperative course was ultimately downward, with continued almost incessant vomiting, accompanied by increasing weakness and incoordination of the left side. She died, June 3, seven weeks after operation.

Necropsy.—This was limited to the head. The brain on section showed a tuberculoma (fig. 20) involving the left dentate nucleus and restiform body projecting as a nodular mass into the dilated fourth ventricle. The left superior cerebellar peduncle was slightly involved by the lesion. The adjacent choroid plexus appeared normal and was easily detached. The meninges and ependyma were not involved. Microscopic examination of a section taken from the dentate nucleus showed the lesion to be a tuberculoma (fig. 21).

Comment.—Death in this case apparently was due to the local effects of the tumor unrelieved by operation rather than to the more common sequence of a terminal invasion of the meninges. The involvement of the vestibular nucleus probably accounted for the excessive

15. This case was cited by Dr. Cushing: Arch. Neurol. & Psychiat. **10**:605 (Dec.) 1923, as case 10.

vomiting that dominated the syndrome. Owing to the fact that a restricted necropsy only was permitted, the primary source and extent of the disease in other parts of the body remain undetermined.

GROUP III (THREE CASES)

In addition to the fourteen cases verified either at operation or necropsy or both, I find three other cases listed in the tumor series as unverified tuberculomas, which should perhaps be included in this report even in the absence of histopathologic identification of the lesion.

Fig. 19 (case 14).—Patient at the height of improvement three weeks after the cerebellar decompression.

CASE 15.—*A cerebral tuberculoma disclosed by a subtemporal decompression in a middle-aged person. Death in three months, without necropsy.*

History.—The patient, E. C., a banker, aged 41, who entered the hospital, Oct. 22, 1914, had been under treatment for pulmonary tuberculosis for four months owing to a low fever and the finding of bacilli in the sputum. There were definite signs in the left apex. A month prior to admission he began having headaches, nausea, vomiting and failing vision. More recently there had been a few jacksonian seizures involving the left hand and the left side of the face.

Physical Examination.—The man was emaciated, and in addition to pulmonary tuberculosis had a marked choked disk with hemorrhages, bilateral abducens weakness, slight left facial palsy and diminished hearing in the left ear.

Operation.—A right subtemporal decompression was performed, October 26, for palliative purposes. It disclosed by chance what was taken to be a large tuberculoma of the temporal lobe. The growth was not removed.

Course.—The patient did surprisingly well and was discharged, November 5, with the pressure symptoms greatly relieved. He died at his home, of generalized tuberculosis, on Feb. 5, 1915. No necropsy was performed.

Fig. 20 (case 14).—Solitary tubercle of dentate nucleus and restiform body projecting into dilated fourth ventricle.

The two following cases are almost duplicates of each other, and the demonstration of tubercle bacilli in the cerebrospinal fluid in the second case before death can be regarded as sufficient verification of the lesion. It may be assumed that the diagnosis of tuberculoma in the other case was correct. If that is so, it represents the only example in the entire series of what may be termed a permanent recovery.

CASE 16.—*Suboccipital operation in a child, aged 9, exposing a tuberculoma histologically unverified. Recovery with six-year survival to date.*

History.—The patient, B. C., a French boy, aged 9, who was admitted to the hospital, Aug. 30, 1920, had become separated from his parents at the outbreak of the World War and was held in occupied territory. He became much undernourished. In August, 1918, he had had an attack of influenza, and a large indolent abscess had subsequently developed in the scalp, which had been incised and had required some weeks to heal. Eight months previously, he began to have frontal

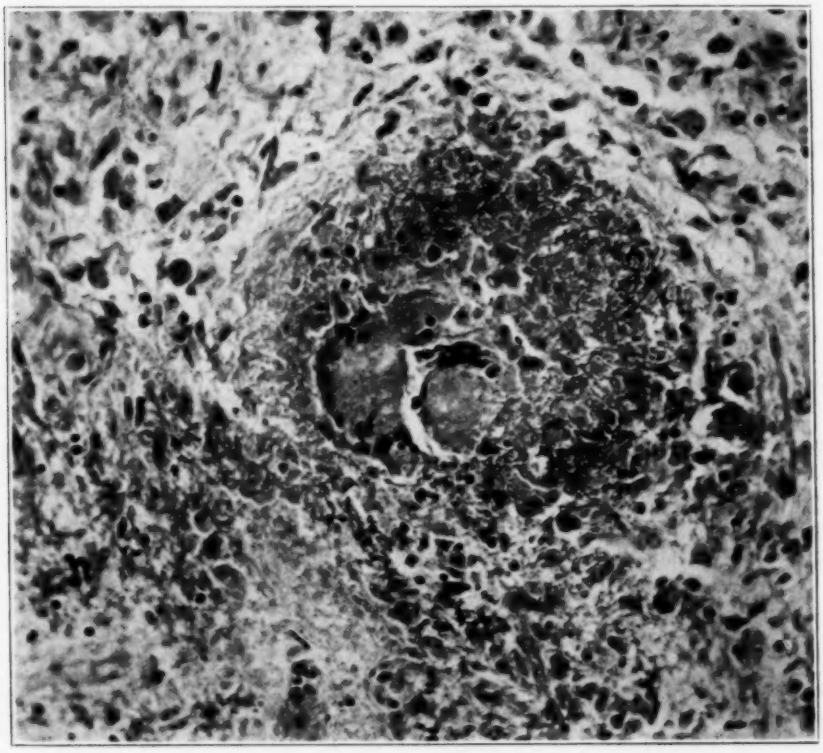

Fig. 21 (case 14).—Section taken near edge of necrotic tissue of the dentate nucleus.

headaches, which were now accompanied by nausea and vomiting, projectile in type. Failing vision was first noted four months previously, and for three months he had had no light perception.

Physical Examination.—The boy was intelligent, cooperative and fairly well nourished, with enlarged cervical glands. The skull gave a "cracked-pot" sound on percussion, evidently from hydrocephalus as confirmed by the roentgen ray. There was secondary optic atrophy from a choked disk. The pupils were dilated and without reaction to light and in accommodation. There were some variable oculomotor palsies with a sustained nystagmus to either side. There was ankle clonus on the left and a bilateral Babinski sign.

Operation.—Though the cerebellar symptoms were inconspicuous, a cerebellar tumor (possibly tuberculoma) was suspected, and consequently on September 14, a suboccipital exploration was made by Dr. Cushing. This revealed, in addition to the hydrocephalus, a dense granulomatous mass attached to the lateral sinus and tentorium in the right upper part of the cerebellar hemisphere. The surrounding cerebellum had the edematous appearance usually seen about solitary tubercles, and the tumor mass was regarded as unquestionably of this nature. Ventricular fluid was injected into a guinea-pig, which died of general peritonitis a month later. As this was not proved to be a tuberculous peritonitis, the exact nature of the tumor remains unverified.

Course.—The child did extremely well. There was perfect healing of the wound. He was given heliotherapy and was discharged on October 17. Subsequent reports up to the present, six years after the operation, state that he has remained well and has been in regular attendance at a school for the blind.

The following case may be looked on as in the nature of a control experiment for the preceding one. The appearance of the lesion was the same, and the second child ultimately died of tuberculous meningitis although the supposed tuberculoma was not histologically verified by necropsy.

CASE 17.—*Suboccipital exploration in a child, aged 8, exposing a tuberculoma histologically unverified. Death from tuberculous meningitis three months later. No necropsy.*

History.—The patient, C. M., a schoolboy, aged 8, of Assyrian parentage, was admitted to the hospital, May 16, 1924, with an essentially negative family and past history. Four months before entrance, he began having headaches with occasional vomiting followed by disturbance of vision and stiffness of the neck with tilting of the head.

Physical Examination.—This was negative with the exception of the neurologic signs. The boy was well nourished and cooperative, invariably lying curled up in bed on the right side. There were: bilateral choked disk of 5 diopters, with hemorrhages and lowered acuity of vision; a large head with separation of sutures; marked suboccipital tenderness; static instability with pronounced cerebellar incoordination and hypotonia of the left extremities, with somewhat inactive reflexes. A few nystagmoid jerks were occasionally observed. The diagnosis was left intracerebellar tumor, probably a glioma.

Operation.—On May 20, a suboccipital exploration was performed by Dr. Cushing, who found in addition to hydrocephalus and a marked pressure cone a resistant granulomatous subcortical mass in the upper part of the left hemisphere adherent to the tentorium. The lesion was exposed by a transcortical incision and was considered to be unquestionably a tuberculoma. The ventricular fluid showed no abnormalities.

Course.—The child made an excellent postoperative recovery; he was given heliotherapy till well pigmented (fig. 22), and was discharged, June 28, free from headaches, and with complete subsidence of the choked disks.

There was difficulty in getting the child suitably placed. In the unfavorable surroundings of his home he did not progress, and a month after discharge was brought back to the hospital with evident tuberculous meningitis. This was verified by ventricular puncture and positive guinea-pig inoculation. He died on August 13; necropsy was refused.

Though the last three cases do not meet the criterion of "verification" established in the clinic, they are recorded for the special reason that case 17 may be an example of permanent recovery from a cerebellar tuberculoma. They serve also as an illustration of the importance of a histopathologic identification of an intracranial growth before one can speak with assurance of its nature and consequently of its prognosis.

COMMENT

As stated in an earlier paragraph, I was led to abandon my original intention of tabulating the surgically treated tuberculomas that have been recorded in the literature. The operative methods employed have

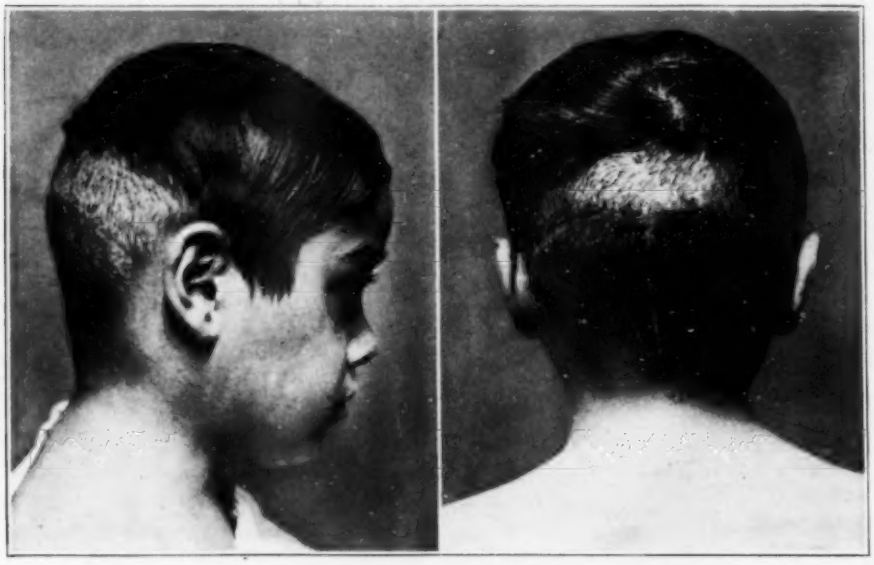

Fig. 22 (case 17).—Child treated by heliotherapy after suboccipital exploration disclosing an unsuspected solitary tubercle.

been so variable, the clinical reports give such insufficient details, and the end-results beyond the period of a few months are so rarely given that a satisfactory analysis of the material is hardly permitted. Rarely has an author reported more than a single case, and naturally the more favorable ones will have found their way into the pages of the current journals. Even so, there are a surprisingly small number of what may be regarded as "cures," though many cases have shown marked temporary benefit, in some instances for a number of months. Of some thirty examples of attempted or successful extirpations of the lesion from the cerebrum, survivals of more than a year are recorded in only five instances. Of twelve recorded operations for subtentorial tubercu-

lomas, there were eight prompt postoperative fatalities and only one patient survived more than a year.

Since the subject is one to which scant attention has been paid in recent years, in spite of the notable advances in the treatment of intracranial tumors of other kinds, the members of the Neurosurgical Society were requested by Dr. Cushing to relate their personal experiences. The responses that have been forwarded show that only fifteen patients have been operated on, four of them for cerebral tuberculomas, eight for cerebellar lesions and the other three not specified. In the majority of cases there has been the usual sequel, with temporary improvement and death in a few weeks or months. However, there have been a few encouraging cases, two of which are reported by Dr. Charles Frazier. One patient, an adult, survived twenty-two months after the removal of a solitary tubercle from the cerebrum.[16] In a still more fortunate instance a solitary tubercle was removed from a child's cerebellum, and though it was bisected in its removal the patient escaped meningitis and is still living and well after eight years. Dr. Charles Elsberg has a patient, also a child, living two and a half years after the incomplete removal of a cerebellar tuberculoma. Dr. Howard Naffziger has an adult patient living more than a year since the extirpation of a deep subcortical tuberculoma of the cerebrum. With these cases I have nothing to compare, since the apparent cure of the local lesion in case 6 was marred by ultimate death from tuberculosis of other organs, and in case 16 the recovery did not follow the removal and absolute identification of the lesion, so that as an example of a cured tuberculoma it is only presumptive.

The experience in case 16 brings up the important question as to the possibility of spontaneous recovery from these lesions if they are left alone. The problem before the neurosurgeon is a complicated one. The necessity of giving relief to pressure symptoms is often urgent, particularly when the lesion is below the tentorium. Whether the likelihood of a spreading meningitis is increased or lessened by an extirpation of the growth, should it prove to be accessible, cannot be definitely stated. It could easily be argued either way from the data at hand.

Tooth endeavors to show in his reports from the National Hospital at Queen Square that in the seven cases in which operation was performed there the patients showed a shorter average survival period than those in which the condition had been left to run its own course, but this at best was a period of only a few months. It must be said, however, that the cases in which operation was not performed were not verified, and Tooth admits that tuberculoma "is often diagnosed but deaths are few." Moreover, his cases were all those of adults, and his surmise that many

16. Frazier, Charles: Tr. Am. Neurol. A., 1921, p. 122.

of the unverified tumor cases in his series which showed a long survival period possibly represented tuberculomas, could hardly be subscribed to at the present day.

However, the surgical outlook for ultimate recovery is poor. When the diagnosis is reasonably assured before operation, judging from the results in this clinic, there would appear to be little justification in doing more than palliate pressure symptoms by proper decompressive measures. When an unsuspected tuberculoma has been exposed in the course of an ordinary exploration for tumor, the temptation will always be strong to remove it, but it would perhaps seem the part of wisdom merely to make room for it and subsequently to build up the patient's resistance to the disease so far as possible by the accepted methods.

SUMMARY

The incidence of tuberculomas of the brain as observed in the Peter Bent Brigham Hospital clinic, where patients of all ages with intracranial tumors are received, is extremely low when contrasted with opinions set forth in the literature of the subject covering the last forty years. Among a series of 1,000 verified tumors recorded up to Oct. 15, 1925, only fourteen, or 1.4 per cent, have been proved to be tuberculous granulomas, whereas around 50 per cent represents the estimate of the earlier writers on the subject.

There are reported in the literature to date certainly not more than fifty cases in which the extirpation of a tuberculoma of the brain has been attempted. Permanent recoveries have been exceedingly few. These recoveries with few exceptions have been after the removal of a cerebral lesion in an adult, whereas solitary tubercle is most commonly found in the cerebellum or brain stem of children.

All but three of the seventeen tuberculomas listed in the surgical records of the Peter Bent Brigham Hospital have occurred in the cerebellum. The lesions as proved at necropsy have been single in the great majority of cases. They were found in patients over 18 in ten instances (seven of them cerebellar); in seven instances (all cerebellar) in patients under 18. In six instances the lesion was surgically extirpated, five times from the cerebellum with death from recurrence of symptoms, usually with a terminal meningitis within three months. Only one patient survived more than a year without local recurrence, this having been an adult with a cerebral tubercle who subsequently died from the effects of an operation for peritoneal tuberculosis. In the other eleven cases palliative measures alone were carried out, and only one of the patients, a child with a subtentorial lesion which was not removed, remains alive and well now six years later.

Regarded from a conservative point of view, it would appear that the operations have been chiefly valuable as palliative procedures, for

most of the patients have for a variable period been immensely benefited. Although the temptation will always be strong to remove an easily accessible solitary tubercle, and though occasional permanent recoveries have been reported, as a curative measure extirpation of the lesion would seem, in most cases, to be of questionable value.

DISCUSSION

Dr. M. Allen Starr, New York: I would like to say a word in explanation of the statistics that Dr. Cushing has quoted as coming from my work.

It is first to be remembered that those statistics were gathered in 1888, which is thirty-eight years ago. The amount of tuberculosis prevalent now is two thirds of what it was then. I have tables showing the prevalence of tuberculosis, or rather the gradual decrease of tuberculosis during the last fifty years, and the percentage of deaths from tuberculosis now recorded is very little over 30 per cent of what it was fifty years ago. So one would not expect as many tumors from tuberculosis now as then.

Secondly, those statistics were gathered largely from publications in England and in Germany and therefore are more applicable to these countries than to the United States.

Dr. Cushing has just said that the Scotch thought that the number of tuberculous tumors in the brain in Scotland was larger than he had observed; so this may be the second explanation for the discrepancy between the statistics gathered in 1888 and those that are available today.

Dr. Morton Prince, Boston: I note that a number of the patients operated on died of tuberculous meningitis and tuberculous peritonitis. Was that due to diffusion of the infection through the operation, and if so, is that one of the dangers?

Dr. Edward W. Taylor, Boston: From the point of view of diagnosis, how much importance do you place on temperature? I recall one case some years ago in which there was no evidence of tuberculosis beyond a certain unexplained rise of temperature. The tumor was sharply localized in the cortex. It was removed and death resulted some weeks later, as is usual, apparently, in these cases. There were various other small tuberculomas near the tumor. In that case the rise in temperature was not explained at the time, and operation was performed without reference to the fact that the condition was tuberculosis, as it turned out to be.

Dr. Ernest Sachs, St. Louis: My experience has been almost similar to that of others. I have found the incidence of tuberculomas very much smaller than I had been led to believe. Seven cases were undoubted tuberculomas. One patient is living after six years, another after four years. The others all died within two months of tuberculous meningitis. Two of that group died shortly after operation, and the diagnosis was made at necropsy.

Of the two surviving patients, one was operated on four years ago, before the Society of Neurological Surgeons, under the impression that he had a tumor, and a localized tuberculous meningitis was found. That boy is still well. I mention that particularly as raising the question of how frequently a patient with tuberculous meningitis may recover. The literature usually states that tuberculous meningitis has a mortality of 100 per cent.

There are two other cases that have been tentatively included in this series, although it is not certain whether they are tuberculous cases of calcified

nodules in the cortex. Histologically, it has been impossible to determine their nature, but I believe that they may be tuberculous processes.

What is your opinion of the type of lesion?

Dr. Harvey Cushing: Dr. Starr's explanation, I think, is correct. The old figures were doubtless compiled largely from European literature, and tuberculosis was prevalent there. In Scotland there is still a great deal of bovine tuberculosis I believe. That is a point which I did not touch on in my presentation of the paper.

Dr. Prince has asked whether the operation in itself serves to infect the spaces. Naturally every effort is made to remove the tumor intact, whether or not it is thought to be tuberculoma. A surgeon may not know he has removed a tuberculoma until afterward. Tuberculous meningitis is slowly progressive, and it is difficult to tell whether it has been advancing before the operation. There is, of course, great likelihood of opening the spinal spaces and of infecting them with tubercle bacilli.

However, I do not think this is a great danger. One of these patients that I spoke of in whom the meninges were known to be infected at the time of the operation lived thirteen months and died of tuberculous peritonitis. It is conceivable that one could disseminate tuberculosis by handling one of these lesions, but that seems unlikely in consideration of experiences with operations for tuberculosis in other parts of the body.

Dr. Taylor spoke about the fever. I think most of these patients have a slight rise in temperature, and the presumptive diagnosis of tuberculoma was made in the majority of the cases. However, there must always be doubt. There are a great many actual tumors in people who have tuberculosis. If every patient who has been or is under suspicion of having tuberculosis of some sort is denied operation on these grounds many actual tumors would not be found. On the same grounds, in order to be consistent, palliative procedures would be withheld; these measures often give great relief even if they are not curative.

Dr. Sachs speaks about one of his long survivals. The case, I presume, was what would be regarded as a tuberculous meningo-encephalitis or tuberculosis en plaque — a lesion in which meninx and cortex are plastered together. These lesions are favorable for treatment, and are somewhat different from a solitary tuberculoma.

Such cases were the ones reported in the olden days when almost all of the operations were performed for lesions in the rolandic area, and mostly in people who had focal epilepsy or partial hemiplegia. It was the easiest place to approach. The majority of all of the modern operations, on the other hand, are for cerebellar tuberculomas, and these would hardly have been touched in those days.

The question Dr. Sachs brought up as to the recoverability of these lesions depends a great deal, I think, on whether they are bovine or not. Doubtless the bovine cases are much more favorable than others; but pathologists do not really attempt to distinguish between human and bovine tuberculosis under the microscope. It is a difficult matter even by culture. I think it is the bovine cases that have an especial tendency to become calcified. Should one be fortunate enough to encounter a bovine tuberculoma which has become calcified and from which the organisms have died out, the conditions would be most favorable for surgical removal should pressure symptoms exist. If they do not, such a lesion, which should show in the roentgenogram, might well enough be let alone.

A MINIMUM SCHEME FOR THE STUDY OF THE MORPHOLOGIC CONSTITUTION IN PSYCHIATRY

WITH REMARKS ON ANTHROPOMETRIC TECHNIC

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The anthropologic studies in psychiatry which were started on the basis of the doctrine of degeneration have yielded little that is of practical value. The methods of physicial anthropology applied to psychiatric problems on the whole have proved fruitless, because the actual formulation of the question, What are the physical signs of the mentally abnormal person? was incorrect. The collection of the enormous number of physical signs of degeneration which adorn the textbooks of psychiatry of the last generation [1] might well be a lesson in the present day revival of constitutional studies in psychiatry.

The body types described by Kretschmer have not yet found adequate definition and delimitation by anthropologic methods. Many of the observations and measurements employed in racial anthropology do not seem to be applicable in the differentiation of smaller nonracial groups of individuals distinguished by pathologic reactions.

The attempt to work out a scheme of clinical anthropology is confronted with special difficulties. It is not possible to carry out an elaborate scheme of observations, measurements and photographs on mental patients. For the present day status of the problems of the physical constitution in psychiatry, it seems much more important to have a small number of data recorded with the maximum of clarity and accuracy than to have observations based on a number of isolated items which are uncorrelated among themselves, and the significance of which is entirely doubtful. A relatively small scheme of anthropologic observations will mean great economy in time, which is the more desirable since the recording of psychiatric data is also time-consuming, if it is going to do justice to the constitutionally significant clinical facts.

It would be desirable to have data concerning the morphologic constitution of relatives of the examined patients. To obtain these data it is

1. Marie, A.: Essai d'anthropologie psychiatrique, in Traité international de psychologie pathologique, Paris, Alcan, 1910, vol. 1.

necessary, however, actually to see and examine the relatives, a procedure which could not be included in this minimum scheme. To record the data of the bodily habitus of a number of relatives from the statement of one examined patient alone is useless, since even normal people with average intelligence are unable, without training, to identify the constitutionally significant types in their family. Recording a great number of such hereditary data from the statements of a few examined patients and using such data for exact biometric studies has been suggested and done, but for the reasons indicated such data cannot be considered significant unless every relative mentioned has been actually examined. For example, we observed and photographed clear pyknic types among relatives of patients from whose statement this would not have been inferred.

The suggestion of the following minimum scheme is based on the study of a relatively small number of individuals (about 150) in which, however, a large number of observations and measurements was made. The scheme is, of course, open to criticism for its many omissions of data which have been used in similar studies, different authors laying stress on different observations and measurements. The measurements of head and skull, for example, have been particularly favored in anthropologic investigation of psychiatric patients, evidently more on the basis of primitive symbolic beliefs than on any well-founded correlations. The guiding principle of this scheme is the recording of data of the morphologic habitus in toto, and not of isolated details which are unrelated among themselves. It is taken for granted that each case investigated shows the record of a physical examination, including a search for definite endocrinologic anomalies (roentgenograms of the sella turcica when indicated). It seems necessary to say in this connection that reliable endocrinologic signs are relatively few, and that the recording of single parts of the body with the addition "of hyper- or hypo- this or that gland type" usually means little.

OUTLINE FOR RECORDING CLINICAL ANTHROPOLOGIC DATA [2]

I. Race and Color.[3]
II. Age.
III. Sex.

2. The purpose of this scheme is to present in condensed form what seem to be the most essential points to be considered. Detailed discussion and evaluation of different methods and types of data are given by Wertheimer and Hesketh: The Significance of the Physical Constitution in Mental Disease, Baltimore, Williams & Wilkins, 1926; Observations and Remarks on the Physical Constitution of Female Psychiatric Patients, Am. J. Psychiat., 1927, vol. 7, no. 3.

3. Data based on this minimum scheme have been gathered so far only for white people.

IV. Occupation.

V. Weight: usual weight and weight variations; if possible, weight curve.

VI. Measurements:

1. Stature.
2. Leg length.
3. Transverse chest diameter.
4. Sagittal chest diameter.
5. Trunk height.
6. Bi-acromial diameter.
7. Bi-iliac diameter.
8. Subcostal angle.

VII. Indexes:

1. Index $= \frac{Leg\ Length \times 10^3}{Transverse\ Chest\ Diameter \times Sagittal\ Chest\ Diameter \times Trunk\ Height} \times 100$

2. Index $= \frac{Bi\text{-}Iliac\ Diameter}{Bi\text{-}Acromial\ Diameter} \times 100$

3. Index $= \frac{Trunk\ Height}{Leg\ Length} \times 100$

VIII. Observations:

1. Face shape, 1 2 3 4.
2. Face profile: angularity.
   hypoplasia of lower jaw or chin.
   regularity or "smoothness" of contour.
3. Conformation of head: round, high, etc.
4. Baldness: regular.
   irregular.
5. Neck: long; medium; short; thick; thin.
6. Trunk profile: spine, 1 2 3 4 5 6.
   abdomen, 1 2 3 4 5 6.
7. General state of nutrition.
8. Skin color, pigmentation, state of vasomotor system.
9. Dysplastic traits:
   (*a*) "Straight-line" or undeveloped profile.
   (*b*) Hands—especially fingers (acromegaloid, etc.).
   (*c*) Disproportion between trunk and limb length, evident from observation alone.
   (*d*) Unusual distribution of fat on trunk.
   (*e*) Unusual arrangement or amount of body hair.
   (*f*) Pubic hair anomalies (feminine distribution in men, etc.).

IX. Brief descriptive formulation of general impression.

REASONS FOR THE CHOICE OF DATA

The reasons for the choice of the data under I to IV are obvious. Weight (V) is regarded as an important constitutional sign. It is for this reason included in a number of constitutional indexes which are omitted from this scheme for reasons that we have previously discussed in detail. The first index listed (VII—1) gives values definitely related to the observation types of Kretschmer. The second index

(VII—2) expresses the pelvic dimension most comparable with shoulder width. It interprets anthropometrically the relationship between shoulder girdle and hips, which is important for the characterization of the trunk outline, indicating eunuchoid disproportion, trapezoid-like trunk of the athletic type, barrel-shaped trunk of the pyknic type (in which shoulder and pelvic breadth approach the same dimension), etc. The third index (VII—3) expresses in a satisfactory form the relationship between the length of the trunk and the limbs, the marked disproportion of which is not without importance. The measurement of leg length alone is sufficient because the relationship between arm length and leg length is constant. The trunk height—leg length index is preferable to the stature—leg length index (often used medically) because stature itself includes leg length. The subcostal angle is a measurement easily taken which seems to be of great constitutional significance. It shows wide variations in persons of different types. This measurement is a simple method of obtaining an indication of typical chest forms and is in a way a barometer of the morphologic constitution. The wide, deep, pyknic chest is accompanied by a wide subcostal angle; the narrow asthenic thorax has a small subcostal angle. The chest circumference is not taken because it is not an entirely accurate measurement, being influenced by fat and posture, as well as by varying factors in technic. Instead, the two diameters are taken.

The data enumerated under "Observations" are selected as the most characteristic features of the body types described by Kretschmer. The shape of the face (VIII—1) and the profile of the trunk (VIII—6) can be conveniently and clearly designated according to the diagrams given in "The Significance of the Physical Constitution in Mental Disease." The recording of body profile has the advantage of giving an indication of posture.

The brief descriptive formulation (IX) records the general impression of the observer. While a subjective element in this is unavoidable, the summing-up of the ensemble of the striking morphologic characteristics of the patient in plain unschematic words seems valuable. This kind of description conveys infinitely more than isolated details with no expressed connection with the general body configuration. At the least, such a short descriptive formulation gives a definite indication of what the patient does not look like. While each observer should have the utmost freedom in expressing the general impression in his own terms, an example may make our meaning clear: "A small, slender, narrowly-built man, with sallow complexion and deep-set eyes, looking much younger than his actual age."

COMMENT ON ANTHROPOMETRIC TECHNIC

The anthropometric technic is described in Martin's textbook,[4] Hrdlička's "Anthropometry"[5] and Sullivan's handbook.[6] All measurements except that of the subcostal angle can be made with Martin's anthropometer. The trunk height referred to in the outline (VI—5) is the vertical distance from the suprasternal notch to the horizontal seat on which the subject is made to sit. The bi-iliac diameter (VI—7) is that taken at the upper outer border of the iliac crests. The leg length (VI—2) is taken from the anterior iliac spine to the lower margin of the internal malleolus.[7] Since the head of the femur cannot be measured directly, Martin[4] gives the accompanying table showing the distances from the anterior iliac spine to the femur head in patients of different heights. This amount is substracted from the anterior iliac spine height in order to obtain the leg length.

Martin's Method of Estimating Distance from Anterior Iliac Spine to Femur Head in Patients of Different Height

Stature up to 130 cm	15 mm.
Stature from 131 to 150 cm	20 mm.
Stature from 151 to 165 cm	30 mm.
Stature from 166 to 175 cm	40 mm.
Stature from 176 cm. and over	50 mm.

The chest diameters are best taken according to the technic described by Hrdlička.[5] The level at which to measure the chest diameters is at nipple-height in man and at the corresponding height of the upper border of the fourth chondrosternal articulation in women. The patient should stand erect with the forearms flexed at about right angles, and the arms lifted forward and upward to about 30 degrees from the trunk. They should be held limply and without tension. This position both relaxes the thoracic muscles and allows easy application of the instrument. In taking the transverse diameter (VI—3) the rod of the instrument lies on the nipple line, with the fixed branch applied to one side of the thorax until it meets the resistance of the ribs. The movable branch is applied repeatedly, with equal pressure, to the opposite side of the thorax during inspiration and expiration, until the medium between the two is determined. The anteroposterior or sagittal diameter

4. Martin, R.: Anthropometrie, Berlin, Julius Springer, 1925.

5. Hrdlička, A.: Anthropometry, Philadelphia, 1925.

6. Sullivan, L. R.: Essentials of Anthropology, New York, American Museum of Natural History, 1923.

7. This distance can be measured directly or the malleolus height (i. e., the distance from the lower margin of the internal malleolus to the floor) can be subtracted from the height of the anterior iliac spine above the floor.

(VI—4) is taken with the fixed branch of the instrument applied to the nipple line, the rod of the instrument to the ribs on the left side, and the movable branch to the posterior part of the thorax. Again, the medium between normal inspiration and expiration is the measurement to be recorded. When both diameters are taken, the instrument is held so that its plane is at right angles to the plane or axis of the thorax.

CONCLUSION

This scheme can be used in a relatively short time and in the wards of a hospital. Its use on a large nunmber of patients will supply data which would be valuable at the present stage of the investigation of the constitution problem in psychiatry.

News and Comment

CENTRAL NEUROPSYCHIATRIC ASSOCIATION MEETING

The fifth annual convention of the Central Neuropsychiatric Association was held at Cincinnati, on October 29 and 30. Clinical presentations, pathologic demonstrations and addresses were made by neurologists and psychiatrists of Cincinnati. The presidential address was delivered by Dr. Albert M. Barrett, Ann Arbor, Mich., on "Neuropsychiatric Interrelationships." Dr. August Wimmer, Professor of Psychiatry and Neurology at the University of Copenhagen, guest of honor, delivered two addresses, on "The Clinical Aspects of Kleptomania" and "Epilepsy in Chronic Encephalitis."

Officers elected for the ensuing year were: president, Dr. Walter D. Shelden; vice president, Dr. Alvin T. Mathers; secretary-treasurer, Dr. Karl A. Menninger; counselor, Dr. Albert M. Barrett.

Abstracts from Current Literature

BRAIN PUNCTURE. E. NEISSER, Med. Klin. **22**:43 (Jan.) 1926.

Quincke was the master who first pointed the way to interference with the brain and spinal cord by means of instruments. In 1904, the author, in conjunction with the late Kurt Pollack, described a method of brain puncture which consists of a quick boring of the soft parts and bone and introducing a hollow needle into the various parts of the brain without opening the skull. The author also described methods by which areas of the cerebrum and cerebellum are safely reached and which are used in tumors, cysts, suppurations, bleedings and effusions. Lichtheim was the first to accept the author's method; by means of it he discovered a brain tumor, and in conjunction with Scholz discovered cerebellar cysts and brought them to operation. Wernicke, also used this method. Then followed a number of surgeons and neurologists who reported experiences with hydrocephalus and tumors; others recognized cysts and tumors, especially Hasler, Unger and Heide, Harry Scholz, Lewandowsky, Stertz, etc. Great progress was made in the Anton Klinik, of which the work of Pfeiffer is best known. By means of brain puncture Pfeiffer recognized all sorts of tumors, discovered *Cysticercus* and established the histologic diagnosis of general paralysis.

Axhausen, in 1913, published a report of the complete results obtained up to that time, which was somewhat as follows: In intracranial bleeding, serous meningitis and meningeal cysts the method is excellent; it proved good in a number of cases of brain abscess; it is not without danger in brain tumor, and should be limited to cases in which clinical symptoms are not sufficient to establish a diagnosis; in all such cases the method should be used. It is of importance in recognizing internal hydrocephalus, and also is excellent for severe cases of subdural hematoma.

Equally good results were reported by Roeper and Binswanger, in 1915. In 1916, Pincus emphasized the dangers of brain puncture. Auerbach and Frankfurt report deaths and wish to supplant brain punctures by small trephinings. The manifold results of operative intervention in brain tumor were discouraging. Interest was then shown in intervention for the relief of pressure by means of callosal and occipital puncture. Then followed such diagnostic methods as encephalography, the methods of Bingel and Dandy. Rindfleisch, Jakobäus and Peter reported favorable results with brain and ventricle punctures and experiences with abscesses and tumors. The greatest advantage of brain puncture is that anything removable is directly discernible and that the various portions of the brain surface may be explored at one time.

In the present technic of brain puncture, the author uses a small bored needle, as the chances of injury to a blood vessel are less than when a thick needle is used. He has practically never used anesthetics, though some men do. Forster, for instance, uses a little ethyl chloride. The author believes that local anesthesia is the best method, as it renders the puncture painless and is least likely to cause the bore hole to be lost.

Briefly, it may be said that brain puncture has resulted in locating tumors of every description—gliomas, cysts and solid endotheliomas, sarcomas, tuberculomas and carcinomas.

Forster, in 1915, wrote: "Phenomenal results are to be obtained by brain puncture as outlined by Neisser and Pollack." Although various authorities, including Oppenheim in his time, Krause and others, have been doubtful, Forster, on the basis of his wide experience, having performed many hundreds of brain punctures, considers brain puncture necessary before referring a patient to a surgeon for trephining. This is true for all tumors except those of the medulla oblongata and pons (which cannot be operated on), and tumors of the cerebellar peduncles (symptoms of which are such that a diagnosis may be made without puncture). In view of his experience, Forster believes that the prejudices toward brain puncture are the result of writing-desk reflections rather than of practical experience.

With only few exceptions a brain puncture is necessary to determine definitely the site of a tumor. Many tumors are difficult to locate — not only those in the remote regions of the brain but also those outside the brain emanating from the meninges. These are the tumors most easily reached by operation, but they also are often found only by means of brain puncture. Forster regularly employs the brain puncture method and rarely fails to locate the process. Also by means of the puncture he is able to determine whether the process is an inoperable glioma or an operable tumor. Tillman and others who successfully use the brain puncture method use it only when localization cannot be determined by other means.

Diagnosis of the type of tumor by brain puncture was established by Pfeiffer, Forster and others. However, in general, one is satisfied to find the tumor substance, when, except in carcinomas, operation is undoubtedly decided on. The most minute particles aid in making a diagnosis and frequently at first examination. However, staining and hardening may be necessary. An operable tumor may wait a few days until this has been done. The general uniformity of the cerebral substance makes a foreign invasion easily recognizable, although the author has punctured the part of a glioma that was fibrous and contained few cells and did not recognize it. The more difficult structure and composition of the cerebellum makes hardening necessary. Forster has pointed out that suspicious material may be found even near the tumor, such as bloody pigment, granular cells, etc.

Brain puncture would, of course, be the preferable method to be used in all tumors if it were entirely free from danger. However, bleeding may occur and therein lies the danger, though this does not occur in the great majority of cases. One death has been reported due to bleeding of the arteria corporis callosi. However, the same clinicians have punctured the same vessels without bleeding. Küttner avoids the posterior cranial fossa on account of the sinus and because of one experience with fatal bleeding, yet others have punctured the sinus without bleeding.

Suffusions are more frequently found at the site of the puncture; they are usually without symptoms or cause only transitory discomfort. The same is true of small bleedings about the puncture canal. To get around this danger from bleeding, it has been argued that a puncture will not cause a healthy brain to bleed — the sicker the brain the greater the danger from bleeding. It has been the author's experience that when bleeding has occurred the prognosis was very poor to begin with, and this should help one to decide whether a puncture should be done or not. Also, for this reason it is important to make the diagnosis early.

To overcome anemia of the brain following puncture, the author uses a hot water bottle at the feet. If an icebag is used it is applied several hours

before the puncture, and when indicated an intravenous injection of salt is given before the puncture. However, the selection of the proper instrument is of greater importance; the thin needle should be slowly inserted and suction should be constant.

If there is no stasis in the transverse sinus and the pressure remains negative or practically so, there will be no bleeding. In case of stasis the author frequently punctures the sinus with a sharp needle, and withdraws blood without injury to the posterior wall.

Puncture is made at the suspected site of the defect. If there is a question as to the frontal lobe or cerebellum, the frontal lobe is examined first. If there are no localizing symptoms, the silent areas are explored, of which the temporal lobe is the one frequently overlooked. If hydrocephalus exists, as a rule there is no tumor of the hemisphere concerned. If there is doubt as to the cerebrum or cerebellum, a ventricle puncture is made or a frontal puncture for examination of the ventricle. If the posterior cranial fossa and cerebellum are involved the operative chances, especially in children, are poor. If it were not for cysts, the author would hesitate to puncture the posterior cranial fossa—but there are some, and finding them leads to the best diagnostic and therapeutic results. The author punctures cautiously and only the surface. Cyst fluid found in the cerebrum indicates tumor, usually an operable glioma, and it is to be regretted that a dural endothelioma should pass unrecognized.

The number of punctures that may be made at one time depends on the case. When it is a question of frontal lobe or posterior cranial fossa, if the author is uncertain of the side, he takes one frontal lobe and the opposite cerebellum and only rarely both sides of the cerebellum at one time. It may be necessary to puncture several times. It is the author's opinion that a surgeon like Cushing, who has reported no less than 1,600 cases of brain tumor without the use of such diagnostic methods as brain puncture and ventriculography, would have still greater results if he adopted the brain puncture method.

The results with brain abscess have been few in the last twenty years. Ear specialists have argued against it. It is not the fear of bleeding but the fear of pus getting into the meninges or ventricle or of a secondary abscess that keeps them back.

Astonishing results are obtained by the use of brain puncture in all sorts of intracranial bleeding, and Kocher writes that when there is the slightest suspicion of intracranial bleeding a brain puncture should be made. Quincke has punctured and withdrawn 150 cc. of subdural blood, bringing about a cure. Melchior says that in such cases the supreme means of diagnosis by brain puncture is greater than in epidural bleedings. Traumatic, pachymeningitic and genuine subdural hematomas are more common than was formerly believed, and are frequently the cause of unconsciousness and death. In subdural bleedings, surgical intervention is considered only when repeated punctures are of no benefit or if the situation remains unclear.

In regard to apoplectic bleedings, the diagnosis is so accurately made neurologically that brain puncture is not necessary. Yet there are unclear cases in which there is old blood that should be removed. Whether this is to be done by puncture or operation is difficult to say. However, if no benefit is derived from puncture, surgical intervention may be resorted to.

In regard to ventricle puncture, it is well known that all forms of hydrocephalus may be diagnosed by brain puncture. However, the author emphasizes that, simple and free from danger as it is to puncture the full ventricle, just so difficult and dangerous is it to puncture an empty or nearly empty ventricle.

One should avoid making a deep puncture but rather puncture at various places. When there is a ventricular discharge, there is danger of prolapse after trephining. A careful emptying of the ventricle sometimes gives relief in brain tumor.

Ventricular discharges which come under the group of serous meningitis are difficult to recognize as such and are rarely investigated, yet it is known that brain punctures have brought about cures in such cases and have confirmed the presence or absence of a tumor.

Forster says that for a brain puncture to be successful it is necessary for the neurologist who has made the local diagnosis to make the puncture. Tillman says that brain puncture is a mighty advance in surgery and has become one of the most important methods of examination. A summary of the literature shows that brain puncture has been of benefit: (1) in subdural bleedings, (2) in all other intracranial bleedings, (3) in the rare cerebellar cysts, (4) in ventricular discharges, (5) in tumors, (6) in abscesses.

MOERSCH, Rochester, Minn.

DISTURBANCES OF MEMORY IN DISEASES OF THE FRONTAL LOBE AND INSULA. HENRY MARCUS, Ztschr. f. d. ges. Neurol. u. Psychiat. **101**:330 (Feb.) 1926.

Since ancient times, the view has been broadcast that a high, broad, arched forehead is an indication of superior intelligence. In ancient sculpture, the gods are represented with towering "olympic" foreheads, and the high, thinker's forehead is encountered in poetry and prose. The truth of the assumption of the significance of an arched forehead in intelligence depends on whether (1) the frontal lobe actually is the seat of the intelligence, and whether (2) a high forehead always contains a highly developed frontal lobe. The latter assumption is in part refuted by the fact that in rickets the frontal lobe can be high and arched, and again by the presence of high, arched foreheads in internal hydrocephalus. In such patients with high foreheads, there is a poorly developed frontal lobe, and one often finds profound idiots with the so-called "olympic" foreheads. The second assumption that the frontal lobe is the seat of the intelligence is still an open question.

Galen, Nemesius and Cortesius believed that the soul was contained in the cerebral spaces or ventricles. Gall emphasized phrenology which, while supported in part by correct observations, did much harm. Our present knowledge of the problem depends in part on embryologic evidence, on physiologic experiments and on clinico-anatomic material. From the standpoint of development, Fechsig and Brodmann have been the chief contributors. Fechsig demonstrated that certain primary centers get their myelin sheaths early, while other secondary centers are sheathed later. He showed that the primary centers contained long tracts which pass from the surface of the brain to the periphery, and that these tracts conduct sensory impressions to the brain and motor impressions from the brain. He called these "projection centers." The other centers which were clothed in myelin sheaths later contained only a few projection fibers but numerous other fibers which connected different brain areas. Fechsig called these "association fibers"; he believed that they subserved higher functions and called the centers in which they occurred "association or memory centers." These were of three types: anterior or frontal, middle or insular and posterior or parieto-occipital. Fechsig was of the opinion that these centers contained the intrinsic thinking or memory organs. The frontal fibers were of most significance because here the greatest number of fibers from

most centers came together. In this connection it is of great interest that the frontal center is clothed in myelin last. The middle or insular center gains its significance because of its connections with the special centers. Brodmann showed by topographic investigations that the frontal lobe can be divided into two parts, a prefrontal portion which is most highly developed in man, and which is of a high structure, and a posterior part having to do with gross, bodily functions. Fechsig's views, therefore, received strong support from Brodmann and were later perfected by Oscar Vogt and Cajal. From an experimental point of view, Bianchi removed large parts of the frontal lobes in apes and found severe disturbances in memory not only for new but also for old impressions. The animal operated on could not avail himself of earlier experiences. Clinically, there is a great deal of material on hand, but it is often difficult and dangerous to draw conclusions. The war gave good opportunity for studying lesions of the frontal lobe, but no clear conclusions are available. Feuchtwanger came to the conclusion that it is not possible absolutely to delimit the seat of psychic disturbances, and that many general stimulation and deficit symptoms are spread over a large part of the brain. In this he is in complete agreement with von Monakow.

Memory is divided into several components, and man has memory centers for speech, writing and reading, as well as for certain higher functions such as music and mathematics. From the researches of Hun, Wilbrand, Cajal, Henschen and others, it is known that there are two types of reception centers, a lower and a higher. In the lower centers, stimuli are received and stored. For example, the lower visual center lies in the calcarine fissure, the higher in the gyrus angularis; the lower auditory center lies in the transverse temporal convolutions and the higher in the lateral aspect of the temporal lobe; the lower olfactory center is situated in the cornu ammonis and the higher in the uncinate gyrus. Cajal warns us, however, that we should not be satisfied with one lower and one higher center; he assumes centers of varying degree. Edgren has localized the music center in the middle portion of the superior left temporal convolution, injury of this region producing amusia. Henschen has localized the mathematical sense in the parietal lobe. The function of the frontal lobe in its anterior portion, the so-called perforated lobe, is still not well known. We know that the posterior part of the left frontal lobe is important for speech since Broca, in 1861, showed that injuries of this region caused loss of speech memories and power of speech. Pierre Marie in recent years places this area in Flechsig's second association center, in the island of Reil and its surroundings. As yet we have no proof that lesions in the island of Reil produce no disturbances in speech.

Marcus reports cases of tumor involving the prefrontal lobe. Disturbances of memory involving this area are of two kinds — for recent and for remote events. In frontal conditions a peculiar disturbance in recent memory is observed, which was first described by Wernicke as loss of ability to appreciate new memory impressions. This was later investigated in various conditions by Kraepelin. Kossakow described it in his syndrome. This disturbance is typical of disease of the frontal lobes and is ushered in by an acute amnesia for new impressions, the latter being retained a few seconds or minutes. In some cases, only the concentration is decreased; in others, the memory of everything before the beginning of the illness is gone, and in still others only the quantity of knowledge is decreased. These patients live in a certain twilight condition. They are often disoriented. Their attention may at times be attracted, and they may comprehend questions and even solve problems.

They show another condition which was first described by Oppenheim and is called "moria" or "Witzelsucht." Although this occurs often in Kossakow's syndrome, there are many cases in which it occurs with no history of alcoholism. These patients show a certain wit which is in great contrast to their status otherwise. Marcus describes also cases with chronic disease of the frontal cortex and with disturbances of remote and recent memory. "It is interesting to see how in a process which passes from the brain surface further into the depth of the cortex, first the memories of more recent times disappear and then the old supply of knowledge. These relations were observed particularly in general paralysis, and have given rise to the theories concerning the successive layers of memory impressions." Marcus reports a case showing involvement of the insula but draws no conclusions as to its functions, except to remark that its position between the speech centers and its close relation to the basal ganglia marks it as important.

ALPERS, Philadelphia.

A BRIEF SURVEY OF AMERICAN PSYCHIATRY, 1914-1924. F. I. WERTHEIMER, State Hosp. Quart. **11**:167 (Feb.) 1926; Allg. Ztschr. f. Psychiat. **81**:442, 1925.

Psychiatry in America, the author thinks, has developed a special and individual stamp. One of its fundamental traits is a consciously pragmatic attitude from which a dynamic conception of mental abnormality becomes possible. The basis for this is a dynamic psychology that is not content with sharply defined diseases and fixed fatalistic constitutions as entities, nor with static elementalistic concepts of descriptive psychology. This point of view has been called by some "psychobiology" and postulates that there is no sharp division between physiologic facts and psychologic facts or mental processes, the latter phenomena beginning at a level when the organism as a whole appears as an integral part of the reaction. Thus the problem of conscious and unconscious is not of primary importance, and the theory itself is not compatible either with a purely introspective psychology or with extreme behaviorism. Chiefly to be considered is the question: Which factors of the inner and outer world are necessary to enable us to repeat or predict a psychologic reaction?

Mentally integrated activity corresponds to a special stage of ontogenetic and phylogenetic development. It is not a specific organization of structure but can be conceived as an organization of function represented by a system of symbolization. This general trend of view has been influenced more or less by the formulations of suggestive psychotherapy and psychanalysis.

Nosologically, the tendency has not been to assume a classification from narrowly defined disease processes but to view psychopathologic phenomena as a complex of facts from which the personality and its immediate reaction to the environment cannot be easily separated. Rather than a disease process of individual organs it is assumed that, on the psychobiologic level, it is the personality that reacts on the basis of phylogenic and acquired habit tendencies with certain reaction types. On the idea that mental disease is an inadaptation to inner or outer needs, the examination of the personality has been furthered. In America, the tendency is not in the direction of creating types so much as it is to consider the abnormalities as the effect of concrete situations in life, instinctive tendencies and habit formation. The practical clinical value of the study of the personality for the estimation of adaptability has been demonstrated in delirium, dementia praecox, morphinism and constitutional psychopathic inferiority. It has been particularly valuable in dementia praecox;

examination has revealed factors, undoubtedly psychogenic, that are modifiable and corrigible. It has been shown that there is an indirect environmental connection between chronic mental disease in a parent and the symptomatologic aspects of a benign psychosis in the offspring. The study of the personality and environment has been found of value in estimating prognosis, such as the distinction between the basic and constitutional make-up of a prepsychotic individual and withdrawal from social intercouse as a protection against inimical surroundings. Catatonic phenomena may be occasioned by excitation of a constitutional "stubbornness." It has been opined that dementia praecox is characterized by regression; the symptoms themselves are regressive or represent an attempt to escape a regressive tendency, such as a transitory manic state. The malignity of the psychosis depends on the depth of the regression and on whether the regression includes archaic material.

In the field of pathology, aplastic changes of the circulatory system in hebephrenic and catatonic states have been found, and atrophy of the gonads has been described in paranoid compensatory reactions. In paranoid dementia praecox states, hyperplastic changes have been found in the tissues. It has been assumed that there is a distinct connection between the psychopathologic mechanism of compensation and the somatic compensatory changes of growth.

Considerable discussion has ensued concerning the affective psychoses. Attempts have been made to relate the stupors, catatonic syndrome and perplexities to the manic-depressive reaction type because of the toxic origin of some stupors and perplexities; also, because the determination of whether a stupor is benign or malignant seemed impossible from observation of the stupor itself. The point is also made that in addition to the actual confusion there is frequently a subjective sense of confusion that could not be caused by a disorder of thinking. These mixed states have not been considered by others as a mixture of formal symptoms but rather "affective oscillations of the personality." Systematic examination in 200 cases of schizophrenia and manic-depressive psychoses revealed that 12 per cent more of the latter showed a situational factor as a precipitating cause. It appears significant, however, that 78.6 per cent of recurring manic-depressive psychoses showed little or no psychogenic origin. These cases did, however, show psychoses in the family history or constitutional abnormalities.

It is significant that the American tendency is to draw no sharp line between the psychoses and neuroses. Some have said that many neuroses have a greater disturbance in the adjustive equilibrium than some conditions deserving the name psychoses. Psychogenic factors are generally recognized together with biologic theories of adaptive deficiencies. It has been said that every soldier could develop a war neurosis under proper circumstances. In epilepsy some have recognized the existence of an epileptic constitution long before the onset of seizures, the defect being more in the instinctive life of the individual. Psychotherapy is advocated, and the prognosis is based more on the mental state than on the frequency of the seizures. In feeblemindedness, psychopathic degenerative states and delinquency, much has been found to be remedied in the environmental influences.

In the field of therapy, aside from psychotherapy, much has been said on the treatment of focal infections. These infections play only a small part in causation, and the numerous operative procedures proposed represent a degeneration of the pragmatic attitude; they have far overreached their goal.

HOWARD, Milwaukee.

ON STRIATE-LIKE DISEASE OF THE CORTEX IN ARTERIOSCLEROSIS. K. NEUBÜRGER, Ztschr. f. d. ges. Neurol. u. Psychiat. **101**:452, 1926.

In addition to the changes described by Alzheimer in the cortex of arteriosclerotic brains, "perivascular gliosis," and senile loss of cells, focal cortical softenings and hemorrhages, and diffuse slight changes in the cell picture, a peculiar striated process affecting certain layers of the cortex frequently occurs. This process corresponds in all important points with the foci of softening in the cortex. Later stages of such striate softenings produce a picture similar to the état vermoulu of Pierre Marie. While such changes are seldom described, they have been known for some time.

Macroscopically, these changes in the cortex can hardly be seen. Foci may be seen in which the structure seems washed out, the color gray-white, with the width somewhat narrowed, and the demarcation from the medulla uncertain. Nissl preparations, however, show the important characteristics. Naked eye examination of sections shows the diseased areas very pale, with the structure practically gone, and the demarcation from the white matter uncertain. This sort of change often involves several convolutions. The calcarine cortex in particular is involved in this process, but other areas of the brain are also affected. Under low magnification, the process is often seen in the gray matter in a practically linear manner. The cortex appears light and poor in cells. The nerve cells are small and shrunken, and the giant cells of the precentral area, though well preserved, are often pale. Loss of ganglion cells affecting chiefly layers III and IV (Brodmann) is seen, but other layers may be involved. Higher magnification shows that while the loss of cells is fairly great it is not extreme. The cells remaining are diseased. Many kinds of nerve cell changes are present, but the dominant type of change is the "ischemic" and "homogeneous" disease described by Spielmeyer, Spatz and Neubürger. The cytoplasm is more or less pale, the nucleus triangular and deeply stained with eccentric or absent nucleolus. At times the nucleolus is pale. Incrustations in the Golgi network often appear in the marginal portions, or the entire cortex shows such incrustation in apparently intact cells. The myelin sheaths are at times unstainable and at times made up of irregular balls or drops. The vessels show severe arteriosclerotic changes. No thrombi are seen. The capillaries and arterioles are narrow. Leukocytes are common around the vessels.

Accordingly, therefore, there occur areas in the cortex which show a striation and layer-like arrangement, and which correspond to the picture known as "coagulation necrosis" (Spielmeyer) or to the early stages of softening before catabolism and organization have taken place. From the point of view of pathogenesis, these areas must be explained on the basis of disturbance in circulation of a nature which will be discussed later. The areas in the cortex are those of ischemic necrosis, and many are fresh areas occurring a few days before death despite their lack of connection with vessels. The underlying pathologic change of the whole process is doubtless the cerebral arteriosclerosis which is seen in various stages, a fact that is further corroborated by the cell changes which occur in circulatory disturbances, the presence of leukocytes in the absence of infection, and further the presence of areas of necrosis in the cortex directly dependent on a vessel. The areas of necrosis in the cortex are fresh, evidence for which is found in the presence of leukocytes which, as shown by Nissl, occur only in the first days of circulatory hindrance.

The exact nature of the changes in the vessels producing such areas of necrosis was not at first clear. Thromboses, emboli, etc., could not be found.

Neubürger was able to explain the changes only on the basis of vasomotor disturbances, apparently angiospasms of the arterioles and capillaries, or in other words, functional vascular disturbances. In this the author is in agreement with Spielmeyer with whom he discussed the possibility and who, in 1925, pointed out the importance of such functional disturbances. The pathologic explanation for an area of necrosis is established if we find a thrombus in an arteriosclerotic vessel in the heart or kidneys, but it is not always possible to find these. Why, then, is it necessary to consider functional vascular disturbances in cerebral arteriosclerosis? That the arteriosclerosis of large arteries does not often extend to the small vessels is well known. In this connection Kraepelin, in agreement with Pick, emphasized the occurrence of vascular spasms in the production of early nervous symptoms in cerebral arteriosclerosis. Arteriosclerosis, moreover, is often connected with angiospasms, as in the early stages of arteriosclerotic gangrene of the extremities in which vascular spasms and degeneration of the vessels are so intimately bound up and for which periarterial sympathectomy is performed to relieve the vascular spasm. Such combinations are further seen in angina pectoris in spasm of the coronary vessels. Neubürger states that spastic constriction occurred in the vessels in the brains he studied and not stasis, the latter being ruled out by the narrow lumen of the vessel. Such spasms caused slowing of the blood stream and diapedesis of leukocytes, as was often seen in the sections. Whooping cough eclampsia in children and cerebral air emboli show similar vascular phenomena. Jacobi and Magnus in an experimental study of air embolism by microscopic examination of the brain demonstrated that vascular spasms occurred in vessels at some distance from the site of the embolus, and indeed demonstrated a general cerebral angiospasm in such cases. Thus, in pertussis eclampsia, we may assume a general angiospasm in addition to the air emboli.

The means of spread of such areas of necrosis is another problem. Many cortical areas show no such necroses, while other areas are particularly affected. The third and fourth layers of the calcarine cortex particularly are affected by the process, or rather it is here that the process shows its greatest accentuation. Whether this is due to a special "Pathoklise" (Vogt) or to a particular lack of resistance of the plasmatic neuroglia and the nervous parenchyma in these layers (Bielschowsky and Jakob) cannot be determined at present, for we know practically nothing of the focal vulnerability of various areas of the cortex.

ALPERS, Philadelphia.

THE PROJECTION OF THE RETINA IN THE PRIMARY OPTIC NEURON IN MONKEYS. B. BROUWER and W. P. C. ZEEMAN, Brain **49**:1 (March) 1926.

Sixteen experimental lesions were performed on the retina of monkeys consisting of enucleation, in one case, of partial lesions in the periphery of the retina and of operations on the upper and lower halves of the macula. Sections of the brain were studied by the Marchi method eighteen days after the operation; some retinal and all the macular lesions were studied histologically by serial section in order to determine the exact extent of the lesion. In studies made of the optic nerves, it was found that the fibers corresponding to the various retinal fields maintain their same relation in the nerve; there is considerable uncertainty about the macular fibers, but they seem to occupy a lateral position. In the chiasm the fibers from the upper half of the retina cross dorsally, while those from the lower part cross ventrally; the macular fibers cross chiefly in the middle of the chiasm. In the optic tracts the pro-

jection is approximately the same in both the crossed and uncrossed tracts. As the primary optic stations are approached, the fibers corresponding to the retinal fields begin to assume the position they occupy in the external geniculate body where, as a result of the degeneration experiments performed on different parts of the retina, it is easily seen that the upper half of the retina is projected medially, the lower laterally. It is further shown that the crossed and uncrossed fibers must lie close to one another in the corpus geniculatum externum.

Degeneration resulting from macular lesions showed that the macular fibers are widely spread throughout the external geniculate body, but they are chiefly found lying in the center and extending dorsally; that is, occupying those parts of the ganglion in which degenerated fibers were not present after lesions of the upper and lower halves of the peripheral retina. From lesions produced on the upper and lower parts of the macula, it is shown that the upper half of the macula is represented in the area adjacent to the upper part of the periphery or medial, and the lower portion of the macula adjacent to the lower sector of the periphery or a lateral position. It is only in the more oral sections of the external geniculate body that this sharp localization is not found. It is therefore evident that the macula is projected between the upper and lower halves of the periphery.

The monocular field of vision is represented in the periphery. In three cases degenerated fibers were seen in the anterior colliculus, and as the fiber endings there were in different areas, it is suggested that in monkeys there is a localization of the different parts of the retina in the cortex of the anterior colliculus. These lesions were always absent after macular lesions, which leads the authors to assume that if fibers for reflex movements of the pupil take origin in the macula, as maintained by some writers, they cannot be myelinated.

In none of the series of cases were degenerated fibers seen in the pulvinar, from which the authors conclude that the pulvinar is not a primary optic station. This is in agreement with the observations of Henschen as lesions of the pulvinar do not produce hemianopia, while the latter results from disease of the external geniculate body. The fact that the superior retinal fibers cross dorsally and those of the lower half of the retina ventrally in the chiasm has clinical importance, since pressure on the dorsal surface may cause defects in the inferior quadrants of the field of vision, and vice versa. These observations correspond to what Henschen and Wilbrand found in man, and should give us a means of distinguishing between tumors of the hypophysis on the one hand, and suprasellar tumors or dilation of the third ventricle on the other.

As to projection of the retinal fibers in the external geniculate ganglion, the authors' observations are not in accord with the views of Henschen, who has always maintained that the dorsal fibers remain dorsal and the ventral fibers ventral, as in the optic nerve and chiasm. In support of the authors' results there is the clinicopathologic study by Winkler who found degeneration in the medial part of the external geniculate body in a case in which there had been previously an inferior quadrantic hemianopia. This view is strengthened by the experimental anatomic work of Minkowski, who found that the fibers coming from the medial part of the external geniculate body proceed to the dorsal half of the optic radiations and terminate dorsally in the occipital cortex, while the fibers issuing from the lateral part of the ganglion terminate ventrally in the occipital lobe.

The later part of this article deals with the projection of the macula on both the primary and the secondary optic centers. Henschen's views of strict localization as opposed to those of von Monokow who insists on the macular fibers being scattered over a large part of the external geniculate body, the optic radiations and occipital lobes, are discussed, along with Holmes' clinical work. The authors conclude from their experiments on the primary optic neurons that the macular fibers have a large distribution in the second optic neurons, as they come into contact with far more cells of the external geniculate body than the fibers arising from the peripheral parts of the retina; hence there must be many fibers in the central optic radiations which conduct stimuli of macular origin. This wide distribution would account for the scarcity of loss of central vision in lesions of the optic radiations and occipital lobe.

STACK, Milwaukee.

TUMORS OF THE CAUDA EQUINA. REPORT OF TWO CASES. JULIAN M. WOLFSOHN and EDMUND J. MOINSEY, J. A. M. A. **86**:1828 (June 12) 1926.

The lateness of diagnosis of tumors of the cauda equina instigated a paper on this subject with a report of two cases. Most patients have been erroneously treated for sciatica, rheumatism, gallbladder disease, appendicitis, neuritis, Pott's disease, lumbago and sacro-iliac disease. Spiller and Frazier found two and three-fifths years elapsing before diagnosis in their cases; Cushing states, "many years' duration"; Elsberg, one and one-half years; Hamill reports a case in which ten years elapsed between onset and proved diagnosis. The explanation for this delay is apparently the obscure and indefinite symptoms. There is usually an acute stage followed by either an intermittent or a chronic stage. A careful history and frequent examination are of help. The syndromes as summed up by Roussy and Lhermitte, which hold true also for acute nontraumatic cases, are: (1) total syndrome beginning at the level of the second lumbar vertebra; (2) middle type — lumbosacral; (3) lower type — purely sacral; (4) hemicaudal; (5) root type.

Once suspicion is directed to the cauda we usually find that roentgenograms and lumbar puncture in the early stages are normal. In the later stages, Froin's syndrome has occurred in punctures below the lesion, but Cushing and Ayer have shown that xanthochromia may exist above the lesion, thus giving lumbar puncture little value. Sometimes the combined cisterna-lumbar puncture of Ayer and Mixter is of value, but this procedure, like that of Queckenstedt, has little localizing value. Air injection in equal amount to that of the fluid withdrawn with production of root pain at the level of the lesion has been described by French authors, but Wolfsohn and Moinsey have had no experience with this method.

The authors believe that the most valuable accessory is the introduction of from 2 to 3 cc. of iodized oil into the cisterna, by Sicard's method, followed by roentgenograms. Two cases are reported with roentgenograms. In case 1, a man, aged 27, complained of ulcer of the left heel. The present illness began fifteen years previously when he had been struck in the lumbar region with a baseball; after this he had sharp pain in the back radiating down both legs. A year later he became bow-legged. After eight months on a Bradford frame for Pott's disease, the left leg was seen to be smaller than the right. Two years after the accident sensory changes began and when he was seen by the authors sensation was absent over the left buttock, back of the left thigh, and the internal surface of the left leg and foot. The ulcer had been caused by a burn of the anesthetic foot two months before he was seen. Physical examination

gave negative results. Neurologic examination showed affection of the fourth and fifth lumbar vertebrae, and the first, second and third left sacral segments. Roentgenograms showed trouble in the region of the fourth and fifth lumbar vertebrae. The Wassermann test and blood examination gave negative results. The spinal fluid was not obtained by lumbar puncture, and cisternal puncture yielded a normal fluid. Roentgenograms taken after the injection of lipiodol showed obstruction at the third lumbar vertebra. Operation yielded a fibroma dorsal to the fourth and fifth lumbar vertebrae, and the patient was soon discharged.

In case 2, a man, aged 34, complained of pain of three years' duration, in the left lower abdominal quadrant and buttocks, and in the lumbar region radiating down the legs to the ankles. He had obstinate constipation and had had dysuria and slight incontinence, but no impotence. Examination gave negative results, except for loss of sensation over the right buttock and the back of the right thigh. A roentgenogram showed six sacral segments. The Wassermann reaction was negative. Lumbar puncture was unsuccessful, but the cisterna yielded a yellow, otherwise normal fluid. A roentgenogram of the lipiodol injection showed complete block at the level of the third lumbar vertebra. A very vascular tumor was found on the right of the cord at the level of the foramina between the fourth and fifth lumbar vertebrae. A cyst was also found on removing the posterior surface of the first and second sacral vertebrae. Bleeding was uncontrollable and the cyst could not be removed; the patient is now receiving roentgen-ray therapy.

Wolfsohn and Moinsey conclude that caudal lesions are more common than supposed and that careful examination and keeping in mind the possibility of caudal lesion will result in earlier diagnoses and that lipiodol injected intrathecally is innocuous and is a valuable adjunct in confirming the diagnosis and in localizing the level of the lesion.

CHAMBERS, Syracuse, N. Y.

ON THE FUNCTION OF THE RIGHT HEMISPHERE OF THE BRAIN IN RELATION TO THE LEFT IN SPEECH, MUSIC AND CALCULATION. S. E. HENSCHEN, Brain **49**:110 (March) 1926.

The author accepts motor aphasia as the result of a lesion of the foot of the left third frontal convolution, or Broca's area. In cases in which partial recovery of speech follows a lesion of Broca's area, Henschen maintains that the right third frontal convolution acts as a substitute. If both left and right frontal gyri are destroyed, speech disappears completely and forever. In every case, however, the right hemisphere shows a manifest inferiority as compared with the left and plays an automatic rôle only; destruction of the left third frontal gyrus seldom interferes with mechanical repetition of words, which is performed by means of the right hemisphere. Word deafness is the result of disease of the first left temporal convolution, and following its destruction there can be a substitution by the same area on the right side, but this power is limited; mechanical or physiologic reproduction of words may be possible, but not psychic comprehension. A person, following destruction of the left temporal lobe, sinks to the level of primitive man; he utters and understands simple words but cannot form combined phrases, which shows that the right temporal lobe is sufficient for the more primitive psychic life.

From the results of his researches the author concludes that there is a special circumscribed graphic center in the foot of the left second frontal convolution. From the five cases reported in the literature there has been no real substitution

of the right hemisphere after isolated lesions in this area. Although one learns to read by word hearing, the faculty of word reading later becomes independent and has a circumscribed center in the angular gyrus, destruction of which always produces word blindiness or inability to read and understand words, syllables and phrases. In destruction of the left angular gyrus there is only a defective substitution by the right, and its function is mechanical only; the person never learns to comprehend psychically the meaning of the word read. The right hemisphere acts best as a substitute for the left in hearing and speaking words; its capacity is less developed as regards reading, and especially for writing. This is due to the fact that the former faculties are more primitive, while the two latter are the result of special training and education; writing indicates the highest evolutionary level of speech.

Music ranks with language as a means of expressing feelings, and most forms, according to the author, are localized in the left hemisphere, the power of singing being most likely localized in the left third frontal convolution in front of Broca's area. In many cases the faculty of singing words is conserved in spite of complete inability to speak a single word. Since the musical faculty is phylogenetically as well as ontogenetically older than speech, its representation is more uniformly distributed over both hemispheres, and hence the right hemisphere can function better when the left is destroyed in music than in speech. The author suggests that the large reserve area of cortical fields in the right hemisphere may be capable of being educated by training and therefore offers a hope of future higher development.

Calculation is widely distributed over the left hemisphere, and since enumeration of figures is a mechanical act, the result of training, the left can be substituted for, to a certain extent, by the right hemisphere. Lesions that cause acalculia, or impossibility of calculation, are often very large and are associated with word blindness and agraphia, or severe visual disturbances. The importance of the left hemisphere is shown by the predominance of the right arm and hand, characteristic of man, and appears among all people from the oldest prehistoric times, and in a child in the first year of life. The author maintains that there is no doubt that right-handedness is inherited from the higher erect animals and is not due exclusively to education, though it is intensified by it.

STACK, Milwaukee.

THE INCIDENCE AND THE PROCESS OF CALCIFICATION IN GLIOMAS OF THE NECK. ARTHUR T. VAN DESSEL, Arch. franco-belges de chir. **28**:845 (Oct.) 1925.

The author considers the incidence and importance of calcification in gliomatous tumors of the cerebrum from two points of view. First, the value of the presence of calcification in the tumor as a means of determining its location by the shadow thrown by the calcified area on the roentgen-ray plates. Second, a comparison between the postoperative survival periods of gliomas that underwent calcification and those which did not showed that the former class is much less rapid in growth and much more benign.

In a series of verified gliomas from Cushing's clinic, where this study was made, calcification within the tumor of sufficient density to be seen on the roentgen-ray plate was noted in forty of 473 cases, about 8 per cent. A more detailed study was made of cases in which roentgenograms were made under the conditions of improved technic afforded by the Potter-Bucky diaphragm. Among 126 cases of verified gliomas in which this method was used, in seventeen

or 13.5 per cent the shadow cast on the roentgen-ray plate by the calcification within the tumor localized its position precisely. Calcification in cerebral tumors is more likely to cast a shadow on the roentgenogram than a similar area in a cerebellar glioma. This is due, in all probability, to the thick petrous portion of the temporal bone which prevents a clear view of the cerebellar region. In comparing a series of cerebral and cerebellar gliomas it was noted that in the latter calcification occurred more rarely probably as a result of the fact that cerebellar tumors, growing in a more confined space, produce symptoms demanding relief relatively early in the course and before they have had time to undergo calcification. Cerebral gliomas, particularly if situated in a silent area of the brain, may grow to a large size and be present for long periods before they excite attention. It is significant that nine of these seventeen gliomas were situated in the frontal lobes.

Histologic classification of gliomatous tumors of the brain shows that the more highly differentiated embryologically the cells forming the tumor, the more benign and slow growing is the neoplasm. And it is among these embryologically highly differentiated tumors that calcification occurs most frequently. The calcium salts are apparently laid down in the bloodvessel walls. This results in slow and finally complete occlusion of the lumen. Minute areas of degeneration are set up as the circulation in the region supplied by the vessel is cut off. These areas coalesce until the glioma undergoes cystic degeneration. The calcium is deposited in the cyst wall. This process of calcification is well illustrated by a number of microphotographs.

From the surgical standpoint, calcified gliomas which have undergone cystic degeneration are extremely favorable. The immediate operative mortality is low. Because these tumors have become cystic, simple evacuation of the fluid may relieve symptoms for long periods. At times these neoplasms are discrete, clean-cut areas easily differentiated from the brain and susceptible of apparently total extirpation. The postoperative survival period may run into many years.

GRANT, Philadelphia.

SODIUM IODIDE THERAPY IN POSTENCEPHALITIC PSYCHOSIS: A PRELIMINARY REPORT ON THE TREATMENT OF SIXTEEN CASES. HERMAN R. CASTRO, State Hosp. Quart. **11**:194 (Feb.) 1926.

Quoting from the literature, the author says that the most significant factor in the mental sequelae after the acute stages have passed is emotional alteration or changes in character. In many instances this becomes noticeable after recovery which seemed to have been reached otherwise. These residuals may be a depressed affect, emotional elevation or irascibility, explosive reactions, stubbornness, etc. In ten of the sixteen cases studied the condition came on immediately after the acute attack; in the other six cases it was observed from one and one-half months to a year later. In fifteen cases the change was characterized by irritability, excitability, stubbornness and marked emotional instability. In two cases stealing habits were added; in two others there were morbid sexual habits; one patient was suicidal, and one had convulsions. The sixteenth patient presented an apathetic, lazy indifferent attitude.

In reviewing the literature, the author finds that treatment has so far proved unsatisfactory. Among therapeutic measures that have been followed with favorable results are: sodium iodide combined with Pregl's solution; iodide with Besredka's typhoid vaccine; a combination of the autolysates of *Bacillus prodigiosus* and staphylococcus. All of these gave some slight results in the

acute but none in the chronic stages. In the parkinsonian and chronic phases, the patients have been treated with sodium iodide, malaria, sodium cacodylate and sodium salicylate, but generally with poor results.

The technic used in the author's series was intravenous injections of sodium iodide twice a week. During the first three months, 100 cc. of a 5 per cent solution was used; this was then changed to 50 cc. of a 10 per cent solution because of poor cooperation and to economize in time. The treatment was usually stopped after the eighteenth injection or after the patient had received 90 Gm. (48 drachms).

Observations are recorded of six patients, three who were paroled and three who had received eighteen or more injections. In no instance was there a sign of iodism; the only complaint was of a saline taste that as a rule lasted from twelve to forty-eight hours. In each case mental improvement was noted. If the patient was indifferent, dull and depressed, he became interested, brighter and happy. If he was irritable and excitable, there was a tendency to become sociable and agreeable. In the case that was complicated with convulsions, the patient became completely stable and the convulsions ceased. As to improvement in the neurologic aspect, the only comment made is that it was less than the mental improvement. The author makes no attempt to explain the improvement, and states that it is too soon to determine the permanence of the improvement or whether the improvement will be progressive.

HOWARD, Milwaukee.

A FURTHER NOTE ON HEPATOLENTICULAR DEGENERATION. STANLEY BARNES and E. WESTON HURST, Brain **49**:36 (March) 1926.

This work is a further investigation of that previously reported by the authors (*Brain* **48**:279 [Sept.] 1925). It consists of a detailed pathologic report of one of the clinical cases of hepatolenticular degeneration previously reported. There were widespread changes in the nervous system, the most prominent being in the frontal lobes and putamen. In the latter there was severe destruction of the small cells, less of the large cells, marked neuronophagia, and a pronounced neuroglial overgrowth, especially in the posterior two thirds; there was abundance of typical Alzheimer cells; myelinated fibers were reduced in numbers; new capillary vessels were abundant. In the globus pallidus the neurons were swollen and without Nissl substance, but there was no notable increase in the number of neuroglia cells. There were mild cellular changes in the caudate nucleus and thalamus. The hypothalamic nuclei showed no important changes and the cerebellar system was normal. The most pronounced pathologic change was in the anterior part of the frontal lobes, with the process having its seat mainly in the white matter forming the prolongations of the centrum ovale into the cerebral gyri. In the early stages there was markedly increased cellularity and new vessel formation; in the later stages there was distinct cavity formation, the white matter consisting mainly of a loose meshed neuroglia reticulum with comparatively few cells and vessels; myelinated fibers were very much degenerated. Only in places was there any considerable injury in the cortex; new vessels were seen growing in from the pia-arachnoid, and the latter was thickened. There was marked cirrhosis of the liver. While pigmentation of the cornea was observed only in the right eye during life, microscopic examination revealed its presence also on the left side. Dark brown pigment granules, closely packed together, occupied the membrane of Descemet. The authors discuss in detail this corneal pigmentation known as

the Kayser-Fleischer zone. All of the sixteen cases of pseudosclerosis reported by Hall in the literature showed corneal pigmentation, and in six of twelve of Wilson's type the Kayser-Fleischer line was present. The authors think that a slit-lamp examination would have revealed more positive cases in the latter group. They further state that no case, regardless of the clinical term applied, in which the zone was present during life failed at necropsy to show cirrhosis of the liver. Chemical tests of the pigment in this case, as well as in Hall's cases, failed to reveal the reactions of any normal body pigment or of any pigment found in the common pathologic conditions. It is the authors' opinion that the liver is always affected before the nervous system, and that there is no essential difference between the recently published cases of pseudosclerosis and of Wilson's progressive lenticular degeneration.

STACK, Milwaukee.

PSYCHIATRIC EXAMINATION OF A CHILD. DIVISION ON PREVENTION OF DELINQUENCY OF THE NATIONAL COMMITTEE FOR MENTAL HYGIENE, State Hosp. Quart. **10**:628 (Aug.) 1925.

The success of a psychiatric examination of a child depends on the establishment of a proper frame of mind in the child, by disabusing his mind of any prejudice, intimidation or factors that would give rise to a defense reaction. Questioning that would give the impression of attempts to elicit confessions of the misbehavior or delinquencies should be avoided. The psychiatric significance of how the child feels about conditions to which he does not adjust, what elements in such situations affect him most and what responsibility he assumes for his difficulty, should be of most importance.

One of the important lines of inquiry is that into family relationships. Such conflicts as a divided household involving a question of the child's loyalty, a step-parent of whom he is jealous or with whom he is incompatible, the death or absence of a parent to which he cannot resign himself, siblings who are brighter or whom he feels are more popular than he, are some of the many situations to be considered. School relationships are a second important field. Examination should reveal whether he likes school and for what reason he began to dislike it and what subjects he likes or dislikes. His attitude toward his teacher and classmates should be determined, whether he feels he is fairly treated and on whom he places the blame for his difficulties. His social relationships, his needs, abilities and aspirations are important, as well as the amount of recreation he gets and what substitutes he has adopted for the lacks in his environment. Is he a leader or does he feel inferior, and if inferior why does he feel so? The dream life is important as the dreams give an insight into desires, fears and conflicts. The attitude toward the opposite sex and knowledge of sex matters should be investigated cautiously to avoid increasing interest or deepening a possible conflict.

During the examination, care should be taken to keep the child's confidence and to guard against his connecting the psychiatrist with any idea of punishment. Questionable points should not be taken for granted if the child refuses responsibility for delinquencies, but information should be received with judicial friendliness. It is necessary to grasp the child's point of view and enter into all his problems with a friendly and sympathetic manner. The summary should include the motivating mechanism, the degree of insight, resources for adjustment and remediable difficulties in the environment.

HOWARD, Milwaukee.

NOTES ON THE RETINA AND LATERAL GENICULATE BODY IN TUPAIA, TARSIUS, NYCTICEBUS AND HAPALE. H. H. WOOLLARD, Brain **49**:77 (March) 1926.

The retina and the lateral geniculate body were studied in a series of closely related animals forming an ascending series and an attempt made to interpret the unfolding of the lateral geniculate body in the terms of the work of Brouwer on retinal localization in that body. In their description of the lateral geniculate body, the authors follow the plan set forth by von Monakow and Minkowski. It consists of a dorsal and ventral portion, the former being the most recent phylogenetically and having a higher degree of differentiation in structure; it gives origin to the optic radiations. The ventral, or older portion phylogenetically, gives origin to the fibers acting on the midbrain.

In *Tupaia*, the lowest animal in this series, the retina shows no differentiated area and the vision is monocular. The lateral geniculate body is notable for the great size of the ventral nucleus, and therefore the midbrain reflexes are relatively important; the dorsal nucleus is small and poorly laminated, thus showing that the cerebral results of visual impression play a small part in the behavior of the animal. In *Nycticebus* likewise the retina is without a differentiated area, but the layers are more thickened. The lateral geniculate body shows a highly specialized ventral nucleus which the author relates to the nocturnal habits of the animal; the dorsal nucleus is small but fairly well laminated. The retina of *Tarsius* presents a highly differentiated area, which can be regarded as a primordium maculae luteae, and there is a corresponding development of the lateral geniculate body. Here the central mass of the dorsal nucleus is well developed, which confirms the experimental conclusions that point to the macula as being represented diffusely about the middle of the lateral geniculate body. The ventral nucleus is much reduced in size with consequently fewer fibers acting on the midbrain centers. This brings *Tarsius* higher in the evolutional scale and foreshadows the dominance of the cerebral aspects of visual influence. The marmoset, the highest animal in this series, exhibits in the retina a close approximation to the final form of the fovea centralis. In the lateral geniculate body there is, in addition to the medial and lateral laminae, a great increase in size and differentiation of the central area. This central mass occupies in the dorsal nucleus a position that almost exactly parallels the localization of the macula in man according to the researches of Rönne. The ventral nucleus and corpus pregeniculatum, which subserve the midbrain centers and not the cortex, are reduced to insignificance.

STACK, Milwaukee.

WHAT HAPPENS TO PATIENTS WITH MENTAL DISEASE DURING THE FIRST YEAR OF HOSPITAL LIFE. HORATIO M. POLLOCK, State Hosp. Quart. **10**:594 (Aug.) 1925.

Because of the inadequacy of ordinary hospital statistics, the author makes this study to show more definitely what happens during the most critical period of the hospital life of patients with mental disease. Two hundred consecutive first admissions of patients with each of the sixteen principal psychosis groups to the New York State Hospitals were studied. The period of the admissions extended from Oct. 1, 1914, to a time prior to our entry into the war, thus eliminating the influence of the war and the unsatisfactory medical and nursing personnel of that time.

The accompanying table summarizes the study. The term "recovered" denotes the condition of a patient who has regained his normal health. The

term "improved" includes also the "much improved" and denotes any degree of mental gain less than "recovered" which warrants the patient's discharge.

Summary of Two Hundred First Admissions in New York State Hospitals

	Per Cent					
	Discharged					
Psychosis	Recovered	Improved	Unimproved	Died	Remaining in Hospital	Readmitted
Senile	0.0	5.5	6.0	62.5	26.0	0.0
With cerebral arteriosclerosis	5.0	12.5	5.5	48.5	28.5	1.5
General paralysis	0.0	8.5	7.0	45.0	39.5	1.5
With cerebral syphilis	7.5	17.0	5.5	37.5	32.5	1.0
With other brain or nervous disease	14.5	14.5	4.5	34.5	32.0	0.0
Alcoholic	51.0	12.0	4.0	6.0	27.0	2.0
With other somatic disease	35.0	7.0	2.5	48.5	7.0	0.0
Manic-depressive	50.0	17.5	2.0	10.0	20.5	2.5
Involution melancholia	15.5	12.5	6.0	22.5	43.5	1.5
Dementia praecox	1.5	21.0	9.5	1.5	66.5	1.5
Paranoia or paranoid conditions	6.0	30.5	11.0	6.5	46.0	2.5
Epileptic	16.0	10.0	4.5	8.0	61.5	3.5
Psychoneuroses and neuroses	31.5	36.5	9.0	2.5	20.5	2.0
With psychopathic personality	41.0	21.0	5.0	2.0	31.0	4.5
With mental deficiency	30.0	13.0	3.0	6.5	47.5	2.0
Undiagnosed	23.5	15.5	11.0	24.0	26.0	0.0
Total	20.5	15.9	6.0	22.9	34.7	1.6

HOWARD, Milwaukee.

ON THE ROTATED OR "CEREBELLAR" POSTURE OF THE HEAD. W. RUSSELL BRAIN, Brain **49**:61 (March) 1926.

The experimental results of various authors, principally Magnus and Muskens, on the rotated posture of the head are reviewed, and sixteen personal cases are presented in which the head assumed rotated positions following different types of lesions. Experimental destruction of one labyrinth or section of the eighth nerve on one side leads to rotation of the head to the affected side. Lesions within the pons, midbrain or forebrain may lead to rotation of the head either to the affected or to the normal side according to their situation. Lesions confined to the cerebellum that do not involve the cerebellar pathways do not lead to the rotated posture of the head. In all clinical cases of internal otitis or of an acoustic nerve tumor, the head was turned to the affected side. This is attributed to interference by these lesions with impulses from the labyrinth on the affected side or, as Magnus contends, the rotated posture of the head and also of the body is due to loss of impulses from the utricular otolith of the affected labyrinth. In five cases lesions of the pons and lesions of the midbrain were found that caused rotation of the head to the normal side.

Muskens' experimental work showed that the head could be rotated either to the homolateral or to the opposite side according to the situation of the lesion, but no satisfactory explanation is offered. Lesions of the cerebellum produced rotation of the head in either direction, and the explanation is advanced that rotation to the affected side is due to pressure on the homolateral eighth nerve, whereas that to the opposite side is due to compression of the pons and medulla on the affected side. The author states that the rotated posture of the head is the result of interference with a tonic reflex in which the extensor muscles of the neck under the influence of impulses from the labyrinths are maintained in a state of tonus adequate to resist the effect of gravity on the head.

STACK, Milwaukee.

HYSTERICAL DYSPHAGIA. HERMAN J. MOERSCH and H. MILTON CONNER, Arch. Otolaryng. **4**:112 (Aug.) 1926.

This disease, first described by Vinson in 1922, is accompanied by pallor and secondary anemia and frequently by enlargement of the spleen. All cases have occurred in women, with an average age of 45, the average duration of symptoms being eight years. Dysphagia begins suddenly, often as a result of nervous shock, and is as pronounced on the first day as at any time during its course. The patients improve, but mental or physical fatigue is followed by relapse. They are unable to take solid food, but in spite of the restricted diet there is seldom loss of weight. The pallor is characteristic, with 50 per cent hemoglobin and normal absolute and differential leukocyte counts. In many cases the teeth had been extracted, and the mucous membranes of the nose and throat were often atrophic and dry, with cracks in the corners of the mouth. Achlorhydria, low color index and changes in the tongue make the anemia resemble a pernicious type. It is a question whether anemia and achlorhydria result from dysphagia or whether dysphagia is caused by esophagitis, glossitis and stomatitis, which in turn cause the anemia. The author does not believe that the anemia is of a pernicious type, but thinks it is possible that the anemia in this condition and pernicious anemia may have a common fundamental deficiency factor. Simple passage of an esophageal sound without stretching cures these patients. "The affection seems to be a functional one with the anemia as a secondary manifestation and it must be differentiated from malignant neoplasm, cicatricial stricture, diverticulum, bulbar palsy, cardiospasm, tuberculosis, syphilis, extra-esophageal tumors pressing on the esophagus and congenital webs of the esophagus which are described in the literature."

HUNTER, Philadelphia.

THE INFLUENCE OF DECOMPRESSION OPERATIONS ON EXPERIMENTALLY PRODUCED PAPILLEDEMA. LOYAL DAVIS, Arch. Surg. **12**:1004 (May) 1926.

The efficacy of subtemporal decompression in the prevention of papilledema due to increased intracranial pressure was tested. Experimental lesions simulating brain tumors were produced in dogs by the insertion into the brain of gelatin capsules containing dried agar agar. As this material absorbed fluid from the brain, it swelled and simulated the expansile growth of a neoplasm. In this way, owing to a localized increase in the content of the intracranial cavity, a definite measurable bilateral papilledema was produced in all cases. The swelling of the disk appeared within from thirty-six to seventy-two hours. On the death of the animal, the presence of edema about the nerve head was proved by microscopic examination. Neurologic symptoms appeared, their character depending on the area into which the agar capsule was inserted. In a second series of animals a generous, well-placed subtemporal decompression was performed before a similar capsule was introduced into the cerebral hemisphere. In five of seven animals no papilledema followed the swelling of the agar. In two in which a much larger amount of agar was introduced, papilledema occurred in spite of the decompression. A similar result was noted when the agar capsule was introduced into the cerebellum to simulate a cerebellar tumor. In the presence of a decompression, no papilledema could be noted. In another series of animals in which the capsule was introduced into the cerebellum, a wide suboccipital craniectomy was simultaneously performed. This procedure was as effective as subtemporal decompression in preventing

the occurrence of a choked disk when an artificial cerebellar tumor mass was produced. The importance of the mechanical factor of increased intracranial pressure in the production of papilledema seems corroborated by these experiments.

GRANT, Philadelphia.

THE NERVOUS SYMPTOMS OF INSULIN HYPOGLYCEMIA IN RABBITS CONTRASTED WITH THE CONVULSIONS INDUCED BY COCAINE. W. RUSSELL BRAIN, Quart. J. Exper. Physiol. **16**:43 (March 18) 1926.

These experiments on rabbits show that the effect of hypoglycemia is primarily on the vestibular apparatus, and that the spells following insulin are not true convulsions with unconsciousness. In his own words: "It is thus apparent that one large group of the symptoms of hypoglycaemia in rabbits, viz., the rotated posture of the head and trunk, and rolling movements around the longitudinal axis to one side, are indistinguishable from symptoms which may be produced experimentally either by unilateral labyrinth extirpation, or by section of the eighth nerve on one side, or by interference with the course of the vestibular impulses within the pons and mid-brain." These phenomena were observed in one human case.

Labyrinth tests were performed on animals with insulin hypoglycemia, and these experiments showed that douching the external ear with cold water (while the rabbits are suffering from insulin hypoglycemia) does not elicit normal responses. Thus, Brain produces direct evidence that the labyrinths are affected by insulin. He concludes: "The rotatory postures and movements of hypoglycaemic rabbits are, then, essentially disturbances of equilibrium not attended by loss of consciousness, diminution of the corneal reflex, or loss of sphincter control. They are to be attributed to a temporary reduction or abolition of afferent impulses from the labyrinths resulting from hypoglycaemia."

In further experiments with cocaine as a convulsant, he shows that accurately timed and controllable convulsions may be produced. These convulsions are described, and seem closely to resemble epileptic convulsions. For experimental work this drug seems to be easier to administer with accuracy than any of the other drugs used by investigators.

COBB, Boston.

IMPROVEMENT OF FACILITIES FOR CARE OF MENTAL PATIENTS PENDING COMMITMENT IN NEW YORK STATE. STANLEY P. DAVIES, State Hosp. Quart. **10**:636 (Aug.) 1925.

One phase of the care of the mentally sick has remained unaffected by modern advances in psychiatry; this is the period of detention in local communities while the patient is being observed or is awaiting proceedings for admission to a state hospital. This local care has too commonly consisted of confinement in a jail cell, a cell in a municipal or county building or unsafe cell-like quarters rented for the purpose. Instead of hospital care, and because of improper medical and nursing care, much unnecessary suffering has been caused and has often so aggravated mental ills that chances of later recovery have been hampered.

To remedy these conditions a special subcommittee of the New York State Committee on Mental Hygiene was appointed to consider what action might be taken. The first step was the preparation and publication of a pamphlet contrasting the right and wrong kinds of temporary care and making suggestions as to the sort of provisions possible in local communities to improve their

facilities. Copies were distributed to health officers, local officials, representative citizens, hospitals, social organizations and others. Copies of this pamphlet, "First Aid for the Mentally Sick," may be had on application to the New York State Committee on Mental Hygiene, 105 East Twenty-Second Street, New York. Definite results have been obtained, and state hospitals serving the territory covered in this special effort report patients in much better condition when received than previously.

The author gives a detailed report of each county visited and the manner in which the facilities were acquired.

HOWARD, Milwaukee.

MELANOPHORES IN DEVELOPING AND ADULT FUNDULUS. ARTHUR SCOTT GILSON, J. Exper. Zool. **45**:415 (July 5) 1926.

Melanophores or black pigment cells of *Fundulus heteroclitus* may be considered to belong to numerous categories of which several play an important part in the color changes of the fish. In the posterior part of the body and tail of the embryo, the melanophores are strikingly metameric in arrangement and in distinct series. This regularity is lost as they increase in number. The melanophores become more and more confined as the tissues become organized in later life. Pigmentation occurs as an intracellular process within certain large mesenchymal cells, beginning before the establishment of the heart beat and continuing at least until after the scales are well formed. The unpigmented melanoblasts show free ameboid activity which is continued after pigmentation during the embryonic life of the fish. At the time of hatching, this freedom of movement is greatly reduced and is absent in the adult. Coincidently with this fixation of the melanophores, there appears the reflex response to light. This response is shown by both deep and superficial melanophores in the young fish, but the activity of the deep melanophores becomes much reduced in the older fish. Study of the pigment granules during melanophore expansion indicates a change from a more viscous to a less viscous or from a gel to a sol condition of the cytoplasm as an initiating step in the activity of form change. The invariability of form of a melanophore in successive expansions, the appearance of recently withdrawn processes and the coordination with the parent cell of pigment masses apparently isolated in recently emptied processes indicate the presence of a fixed bounding membrane.

WYMAN, Boston.

THE RELATION OF STATE HOSPITAL CLINICS TO THE MENTAL HEALTH OF SCHOOL CHILDREN. WILLIAM A. HOWE, State Hosp. Quart. **10**:615 (Aug.) 1925.

Since the adoption of the state mandatory school medical inspection law, there has been a marked increase in the number of children found with disturbances of the nervous system. This law applies to approximately 800,000 children; the examination during the school year, 1923-1924, showed an increase of 9,036 patients with disorders of the nervous system over those found during the school year 1915-1916. The author feels that many children with these diseases are still not recognized as such because medical examiners for the most part are unfamiliar with nervous and mental disorders. As a result, children are allowed to develop a true psychosis or psychoneurosis because the liability to those conditions has not been recognized.

It is suggested that responsibility for improving this situation rests with psychiatrists and particularly with the medical staffs of the state hospitals. The mental hygiene clinics already instituted are commended, but additional

measures are recommended: training in psychiatry for the medical profession in general; stimulation of interest in mental hygiene on the part of parents, teachers and other interested agencies. The latter might be accomplished by sending information to those interested and by establishing clinical centers at hospitals to which patients might be sent for diagnostic and directory purposes.

The fact that there are a large number of neurotic teachers is responsible for the requirement, beginning September 1, of a certificate of physical and mental health of all matriculants in the state normal schools.

HOWARD, Milwaukee.

PROGNOSIS IN DISSEMINATED SCLEROSIS. JAMES TAYLOR, Brain **49**:105 (March) 1926.

The author cites a number of cases of this disease in which there were definite remissions over a period of years, one of which was fatal eighteen months after the onset, and one, the only case known to him, in which there was complete recovery. This patient, a woman, aged 31, had definite pyramidal tract signs, paresthesias and nystagmus in 1911; with rest these symptoms cleared up, and there has since been no recurrence of them or abnormal observations. As to etiology, the author believes that a poison gains access to the body from the outside. It is assumed that in cases similar to those described the toxin produced is liable to periodic release under conditions which favor this, like some slight injury or illness, and that then the natural antitoxins of the blood are called out and the further release of the toxins is prevented or their activity perhaps mitigated or controlled. If these natural antitoxins are not sufficient in quantity or are defective in quality, a progressive disease may be established, its progress not remaining uniform. The author believes that overwork or strain of a definite character favors the development of disseminated sclerosis, and he stresses the point of absolute rest in the early stages of the disease as having a favorable influence on the course of the illness.

STACK, Milwaukee.

HYPOGLYCEMIA AND CONVULSIONS. EDITORIAL, J. A. M. A. **86**:1351 (May 1) 1926.

The advent of insulin has awakened an interest in hypoglycemia with its tendency to cause characteristic symptoms of menacing severity. The administration of sugar has an antidotal effect in the convulsive seizures of hypoglycemia, whether these result from overdosage of insulin or from extirpation of the liver. The significance of hypoglycemia appears to be exemplified in certain obscure convulsions in children, particularly at the outset of acute infections. Josephs has furnished illustrations of convulsive attacks after short periods of fasting—often scarcely more than over night. In healthy children such an alteration produces a decrement of only 15 mg. or less, while febrile patients exhibit hypoglycemia of convulsive levels. Fever, according to Josephs, is not necessary for the production of convulsions. Its significance is twofold: (1) anorexia occurs with it; (2) hypothetically, the children seem unable to mobilize carbohydrate as rapidly as needed, causing a fall of its amount in the blood. It may be that the available carbohydrate is more rapidly used up or that the child's nervous system is rendered more unstable by fever, and the lowered concentration of blood sugar provokes a convulsion which could not occur at normal temperature.

CHAMBERS, Syracuse, N. Y.

THE CHILDREN'S UNIT AT KING'S PARK STATE HOSPITAL. HARRIET A. ROBESON, State Hosp. Quart. **10**:578 (Aug.) 1925.

To obviate the unsatisfactory method of housing children in the same wards with adult patients, two cottages were opened for children under 16, one for boys and one for girls. The cottages were intended for children with all types of mental disease, and during the first year there were fifty-eight admissions. Forty-one of the patients had a history of epidemic encephalitis; the other seventeen included diagnoses of dementia praecox, manic-depressive psychosis, mental deficiency and brain tumor.

The cottages were cheerful and homelike, all guards being taken from the windows, etc. The problem at once resolved itself into one of emotional control and of retraining the children with a habit-training schedule. This schedule was a well balanced one of occupational therapy, academic work, personal toilet and play. After a very trying initial period of adjustment, results were slowly obtained. Physical improvement was prompt. Emotional instability, behavior disorders and pernicious sex habits responded more slowly but gratifyingly. The most favorable results were obtained in the behavior disorders following epidemic encephalitis. As a whole, the children responded better to academic work than to occupational therapy.

HOWARD, Milwaukee.

THE EFFECT OF HETEROPLASTIC GRAFTS OF THE SPINAL CORD ON THE DEVELOPMENT OF THE LIMB OF AMBLYSTOMA. H. L. WIEMAN, J. Exper. Zool. **45**:335 (May 5) 1926.

Experiments were undertaken to test the effects of reciprocal innervation on the development of the limb bud in two species of salamanders, *Amblystoma tigrinum* and a smaller species *Amblystoma punctatum*, by leaving the limb buds undisturbed, but interchanging the limb level of the cord, thus providing conditions for a tigrinum limb to receive its nerve supply from a punctatum spinal segment and vice versa. Segments of the spinal cord coextensive with somites 3, 4 and 5 were interchanged in the two species in early embryos. A structural and functional union between the grafts and the nervous tissues of the hosts occurred. Spinal nerves grew out from the grafted segments to form a brachial plexus. The operation produced no perceptible effect on the rate and manner of development of the limb in early stages of development, and in one case at least up to the time of metamorphosis. The effect of foreign innervation on the development of the limb was nil. The forelimb rudiment of *Amblystoma* is a self-differentiating system. Function is not established with the ingrowth of nerve fibers but depends on the formation of proper connections within the central nervous system.

WYMAN, Boston.

A COMPARATIVE METHOD FOR DEMONSTRATION OF NORMAL DEVELOPMENT IN INFANCY. ARNOLD GESELL, J. A. M. A. **86**:1277 (April 24) 1926.

The author believes that medical students should become more systematically familiar with the normal. Accordingly, he reports one session devoted to the clinical demonstration of two pairs of normal infants, aged 4 and 6, and 6 and 9 months, respectively. The method was to note the spontaneous behavior of the babies as they reacted to the various tests. Dr. Gesell believes that an infant is as old as his behavior, and the results are interpreted in this light. Various tests are used such as lifting and placing side by side to note sitting posture, placing in dorsal position, placing in prone position, dangling a red

ring in front of babies, dropping a spoon and other tests. There are comments and interpretations of the results of each test. The author calls attention to the fact that there are differences in behavior of these infants not only in intelligence but also in personality make-up. This brings out the necessity of such a comparative approach to the problem of developmental diagnosis. The pediatrician must then take a critical interest in such norms of development. Development may be studied from a functional standpoint — behavior — as well as from a standpoint of anatomy and physiology.

CHAMBERS, Syracuse, N. Y.

PSYCHIATRIC PROBLEMS IN THE CARE AND MANAGEMENT OF DELINQUENT CHILDREN IN THE STATE OF NEW YORK. V. C. BRANHAM, State Hosp. Quart. **10**:609 (Aug.) 1925.

Fundamentally, the treatment of the delinquent child should be psychiatric in character. He should be considered to be what he is and to react as he does because of certain physical, emotional and instinctive factors within him in their relationship to his heredity and environment. At present the psychiatric phase of supervision is inadequate for several reasons. Judiciary disposition of such children is ill-advised because of the lack of knowledge of the children and of the various institutions maintained for them. The institutions are at fault because some do not realize the importance of psychiatric supervision; others lack funds to carry on such work, and frequently the practice of the penal code is pursued rather than an effort to make an intelligent study of the child. There are twenty institutions conducted for delinquent children; none of them operate to full capacity owing, probably, to lax probationary methods. The author feels that when the repressive, disciplinary methods are replaced by a psychiatric service that will make a study of each individual, classifying him as to his own intellectual level and mechanical aptitude, the prognosis of the delinquent child will become more favorable.

HOWARD, Milwaukee.

WHAT CONSTITUTES MENTAL HEALTH IN CHILDREN? EDWARD A. STRECKER, Am. J. Dis. Child. **32**:409 (Sept.) 1926.

In the introduction to this article, Strecker pointedly observes, "The normal child has been called a polymorphous pervert, and has been accused of playing the primary rôle in the grim tragedy of Oedipus Rex, and still he has survived. It is unlikely that anything I may say can injure his somewhat spotted reputation." The qualities of a child of normal health and mind are best stated in the words of the author: "The ability and the desire to move, a certain readiness and willingness to imitate, some response to suggestion, a strong leaven of curiosity, an appreciable love of power, a dash of savagery and a seasoning of romancing. There should be intelligence enough to bring out these traits; enough emotional virility to impress the lessons which they teach, and moral judgment to act as a kind of partial check. The marked diminution of these characteristics is a much safer measure of a sick mind than is their excess. The final measuring rod is the ability to pass through the critical phases and make adjustments somewhere within the average time limits."

VONDERAHE, Cincinnati.

AN EXPERIMENTAL STUDY OF TONUS IN SKELETAL MUSCLES AS RELATED TO THE SYMPATHETIC NERVOUS SYSTEM. A. KUNTZ and A. H. KERPER, Am. J. Physiol. **76**:121 (March) 1926.

Extirpation of the sympathetic nerve supply to the leg in dogs, the authors found, produced under anesthesia definite flaccidity and diminution in extensor rigidity although, under ordinary circumstances, no apparent effect was detectable to gross inspection and palpation. Studies were made also of quadriceps tonus curves in cats and dogs before and after unilateral ablation of the lumbar sympathetics and before and after ablation of the cerebellum and vestibular nuclei. Sympathetic section was followed by pronounced hypotonic phenomena and cerebellar deprivation by a similar change but of milder degree. After sympathetic section it was found further that muscle so denervated was not additionally affected as to tonic status by nicotine or cerebellar and vestibular deprivation. The conclusion is reached that while the cerebellar and vestibular components appear to be really important in relation to normal muscle and postural tonus, on the basis probably of direct fiber connection, nevertheless the actual mediation of such function would seem to be accomplished through the mechanism of the sympathic innervation as such.

RAPHAEL, Ann Arbor, Mich.

STUDIES IN ANIMAL AGGREGATIONS: CAUSES AND EFFECTS OF BUNCHING IN LAND ISOPODS. W. C. ALLEE, J. Exper. Zool. **45**:255 (May 5) 1926.

Land isopods form aggregations when exposed to drying conditions but much less readily when the substratum is moist. Bunching behavior is exhibited in both hibernation and estivation and is affected by temperature and illumination. Bunching is effected by the positive thigmotropism and chemotropism of the isopods, with only slight evidence of mutual attraction as such. Normally, bunching takes place about one of the most quiet isopods in the least stimulating part of the container, which is reached by random movement. Bunched isopods take up or lose water less rapidly than isolated individuals when they are out of moisture equilibrium with their environment. Recently formed bunches respire less rapidly than recently isolated isopods per unit of weight. After both have stood for some time, the conditions are reversed. The bunching behavior is thus shown to be advantageous to the individual and probably represents an early stage in social development. Apparently the first step toward social life in lower animals is the appearance of tolerance for other animals in a limited space. The second step is taken when the aggregation comes to serve the individual members. The isopods have reached this stage.

WYMAN, Boston.

THE LOGIC OF SUBLIMATION: A CRITICISM. JAMES L. MURSELL, J. Abnorm. Psychol. **21**:75 (April) 1926.

The idea expressed in sublimation may be correct, but the uses to which the word is put are objectionable and the mechanism of its expression as claimed by the psychanalyst fails to prove anything. There is sublimation, but its freudian foundation lacks acceptability. The free-association and recapitulation approaches to an understanding of the evolving of sublimation are not of proved validity. It is claimed that sublimation is worked out in the subconscious. The author seems to feel that this claim is made by psychanalysts to avoid being forced to produce material or visible proof of the assertion

that sublimation does occur. His belief in his proofs of the failure of the freudian conception of sublimation is at variance with his disbelief in the evidence advanced by the psychanalyst, and yet they seem to be made of practically the same stuff. He calls attention to some of the weaknesses of the psychanalytic practices and theory, and asks proof when the proof is certain to be inadequate to one who reads it in a book.

HAMILL, Chicago.

A STUDY OF THE EFFECT OF MODERATE DOSES OF ALCOHOL ON THE GROWTH AND BEHAVIOR OF THE RAT. CURT P. RICHTER, J. Exper. Zool. **44**:397 (April 5) 1926.

Alcohol was given to rats in drinking water continuously over long periods of time (from three to seven months), so that chronic rather than acute effects were produced. It was found that the white rat is able to utilize an 8 to 16 per cent solution of alcohol as its steady fluid supply, replacing isodynamic quantities of food without intoxication or habituation effects, but with a definite decrease in spontaneous activity. The running activity was less in the experimental animals than in the controls. They were as alert as the control litter mates. It was shown that the ability to digest such large quantities of alcohol without serious effects was due to its high rate of metabolism. Rats that took alcohol ate from 16.9 to 35.6 per cent less than the controls. They grew just as rapidly, however, and reached the same body weight at maturity as the controls. Alcohol not only replaces isodynamic quantities of food in maintaining energy balance but it is also used for growth and development.

WYMAN, Boston.

THE INTERPRETATION OF SPEECH BY TACTUAL AND VISUAL IMPRESSION. ROBERT H. GAULT, Arch. Otolaryng. **4**:228 (Sept.) 1926.

For patients totally deaf, the spoken voice is amplified and the vibrations are transmitted by a special apparatus to the skin. The speed, intensity, uniformity, interval, etc., of the varying vibrations create characteristic sensations, which the patient learns to interpret. Illustrations are given in which patients with little training were able to understand a story and grasp many of the words correctly. They were able to sense the pauses between words and phrases. They frequently perceived the vowel correctly and from the context were able to fill in words that they did not feel through the apparatus. This new method bids fair to be an important addition for training the deaf. It has already been an aid when combined with lip reading. By giving the patient a sense of rhythm, emphasis, accent and tempo, increasing their efficiency to 100 per cent, many words that cannot be distinguished visually on the lips can be discriminated by their feel.

HUNTER, Philadelphia.

GENETIC ANALYSIS OF THE TEMPERAMENT OF RATS. MARY P. SADOVNIKOVA-KOLTZOVA, J. Exper. Zool. **45**:301 (May 5) 1926.

A genetic analysis of the abilities of families of rats was made by testing their work in the Hampton Court maze which has a correct path from the entrance to the center 127 meters long with seven sideways. One hundred and twenty-three rats (obtained from crossing a wild gray with a laboratory white rat) showed a great variety of abilities. The curve of variation shows three chief peaks. Between the abilities of males and females, no real differ-

ence is to be observed. There exists a real correlation between the abilities of parents and those of children and also between the abilities of individuals of the same brood. The teaching of parents does not influence the abilities of the offspring. The abilities of the rats observed belong to the domain of chemico-psychic abilities. It is suggested that a group of three genes is present: (*a*) the gene of general activity, (*b*) the gene of the emotion and (*c*) the gene of the seeking instinct.

WYMAN, Boston.

ADDITIONAL EVIDENCE FAVORING THE NORMAL EXISTENCE OF THE LATERAL APERTURES OF THE FOURTH VENTRICLE IN MAN. A. T. RASMUSSEN, Anat. Record **33**:179 (July 25) 1926.

From a series of brain gathered in a dissecting room during ten years, the author has been able to demonstrate the actual presence of the lateral apertures of the fourth ventricle without complicating manipulative procedures. Cadavers suspended by the head frequently showed, due to fluid loss, shrinkage of the brain and its coverings. The slow retraction of the leptomeninges and the shrinkage of the ninth and tenth cranial nerves resulted in exaggeration of the normally small apertures in the lateral portion of the fourth ventricle, so that their presence was obvious. No contribution is made regarding the medial aperture.

WOLFF, Boston.

BRANCHIOMERISM AND THE THEORY OF HEAD SEGMENTATION. B. F. KINGSBURY, J. Morphol. **42**:83 (June 5) 1926.

A critical review of the developmental evidence shows that the branchial pouches are formed in cephalocaudal sequence subsequently to the segmentation of the dorsal mesoderm. The pouches interrupt a continuous sheet of mesoderm to form the branchial arches. The arches when formed do not correspond topographically to the dorsal somites. Branchiomerism does not, therefore, coincide with somitic metamerism. The branchial structures do not support the theory of head segmentation. The trigeminus, facialis, glossopharyngeus and vagus cannot be regarded as segmental nerves. There is no evidence that branchial pouches or arches have been elided from the series. The problem of meristic homology is briefly discussed.

WYMAN, Boston.

ANGIONEUROTIC EDEMA. REPORT OF A CASE WITH NECROPSY FINDINGS. ISABEL WASAN, J. A. M. A. **86**:1332 (May 1) 1926.

Stimulated by the fact that there are so few postmortem records of reported cases of angioneurotic edema with fatal termination, the author reports a case, with necropsy, which in brief was extreme edema of the respiratory tract with a mild and variable type of cellular reaction. The lung in some sections showed early inflammation. In other sections the changes in the lungs and pharynx more closely resembled a protein sensitization.

CHAMBERS, Syracuse, N. Y.

Society Transactions

PARIS NEUROLOGICAL SOCIETY

February, 1926

Abstr. from Rev. Neurol. **1**:165, 1926

PERSISTENT CHOREO-ATHETOSIS FOLLOWING AN ELECTRIC SHOCK. DR. CROUZON.

The man had received a shock of 1,500 volts six years previously and since had had pronounced choreo-athetotic movements in the right upper limb and to a less degree in the right lower limb. In addition there were tics or spasms of the face, nose and pharynx. The interest in the case lies in the chronicity and constancy of the condition, which today is exactly that of three years ago. All questions of simulation and of pension neurosis must be set aside, because the man is already receiving a pension, and even close watching discloses no change. Nothing has occurred that throws doubt on the organic nature of the disturbance.

SCIATIC PARALYSIS FOLLOWING DYSTOCIA. DRS. CROUZON, CASTERAN and CHRISTOPHE.

Following a difficult confinement in which forceps were used, a dead child was delivered, weighing 13 pounds (5.9 Kg.). There was brisk postpartum hemorrhage. During the application of the forceps, the patient cried out on account of sharp, extremely violent pain with tearing sensations in both feet and in the lower third of the legs. These pains persisted during the next five or six days. Mild puerperal infection followed. After three weeks the patient was up, but found it impossible to move her legs and feet. The left leg rapidly recovered, but the right remained paralyzed for a considerable time. The second pregnancy resulted in normal delivery. Examination four years after the accident revealed mild atrophy of the muscles of the right leg, and some dropfoot with hypotonia and abolition of both Achilles' reflexes. The only subjective complaints were of formication in the buttocks and foot. Objectively, no disturbance was noted. Pressure on the muscles was painful. The Lasègue sign was positive on the right side. Electrical examination showed hypo-excitability on both sides in the supply of the external popliteal nerve. The localization of the disturbance in the peripheral supply of the external popliteal branch of the nerve is common in obstetric paralysis, probably the result of compression of the fibers destined for this nerve at the superior strait. These fibers can be traced up through the sacral plexus and lie anteriorly; therefore, they are more exposed to the pressure of the descending head.

HYPERTONIA AND CONTRACTURE IN GENERAL PARALYSIS (STRIATE SYNDROME). DR. TINEL.

Because of the wide distribution of the pathologic process in general paralysis, this disease is of little value in localization. However, particularly in the late stages, in some cases hypertonia develops which greatly resembles decerebrate rigidity. In a number of these cases, lesions have been described

in the basal ganglia, thalamus, red nucleus, etc. The striatum is always involved, however. Two cases are described showing marked spasticity with athetotic movements and severe contracture of the hands together with other signs of general paralysis.

SCIATIC PARALYSIS FOLLOWING TRAUMATIC DISLOCATION OF THE HIP. DR. CHRISTOPHE.

The patient was knocked down by a truck; a few minutes after the shock he felt a violent pain on the posterior aspect of the thigh and right leg. The limb was completely useless and was fixed in a position of extension and internal rotation. The dislocation of the hip was reduced the next day. Following this, flexion of the leg and the thigh reappeared, but no movements of the foot were possible. Examination a month later showed marked hypotonia of the sciatic distribution, flaccid paralysis, abolition of the Achilles reflex, and slight wasting of the muscles. Vasomotor and trophic disorders were seen in the foot and toe with some edematous infiltration. These disorders are regressing, but there is still some pain and tingling in the limb, and objectively there is anesthesia of the foot except for the internal border, with hypesthesia in the leg. Reduction in pain sensation is present over a similar area. The sense of position is imperfect in the toes. Vibratory sensibility is diminished over the external malleolus. Electrical examination shows practically no reaction of degeneration of the muscles.

FREEMAN, Washington, D. C.

May 27, 1926

Abst. from Rev. Neurol. **1**:929, 1926

A NEW CASE OF INTRACEREBRAL SCLEROSIS (SCHILDER'S DISEASE). DRS. FOIX, BARIÉTY, BARUK and JULIEN MARIE.

The case reported was subacute in evolution with a long stationary period of ten years, and terminated in idiocy, blindness and intense rigidity with epileptiform attacks. Necropsy showed symmetrical destruction of the central portion of the hemispheres affecting the occipital lobes, in this case also the frontal. This case was reported on account of the latent period and because of the mild symptomatology first noted.

MYOCLONIC SYNDROME OF THE TEGMENTUM PONTIS. DRS. FOIX, CHAVANY and HILLEMAND.

This is the report of a clinicopathologic investigation of four cases showing clinically the so-called nystagmus of the palate, a variety of myoclonia. There were other signs pointing to injury of the brain stem, such as paralysis of associated lateral movements of the eyes, hemitremor and vestibular inexcitability. In some cases there was paresis or paralysis of the cranial nerves, especially the fifth, sixth and seventh. Palatal nystagmus is a curious symptom described by Spencer, in 1886. It consists of unilateral or bilateral myoclonic movements of the palate and is often associated with similar movements of the face and jaw. The rhythm runs from about 60 to 120 a minute, is constant for the person and is regular. Necropsy in the four cases showed various areas of destruction in the tegmentum of the pons; but they all showed destruc-

tion of the central fasciculus, so the authors believe that the syndrome is caused by destruction of this tract. It is followed anatomically by rather marked hypertrophic degenerative changes in the olivary nucleus of the same side.

MULTIPLE TUMORS OF THE LATERAL VENTRICLE [TUBEROUS SCLEROSIS]. DRS. JUMENTIÉ and BARBEAU.

A girl, aged 8, with idiocy and epilepsy, showed further regression and died in status epilepticus. Section through the brain disclosed many small glial tumors in the walls of the lateral ventricles. These consisted of cells of various sizes with fibrosis, concretions and some large hyaloid rounded cells with vesicular nuclei and sharply stained nucleoli. The case is obviously one of tuberous sclerosis as described by Bourneville, but in the article is not mentioned as such.

THE NUCLEUS MESENCEPHALICUS TRIGEMINI. DR. NICOLESCO.

Comparative anatomy furnishes interesting data on the structure of the mesencephalic nucleus of the trigeminus. The vesicular cells belong to an ancient phylogenetic formation. The nucleus is important in birds and mammals. In birds there is an intimate connection between the nucleus and the optic lobes together with the contiguous mesencephalic structures. In mammals the cells are in close relationship with the anterior and posterior corpora quadrigemina; probably they have the function of relaying proprioceptive sensibility.

PARKINSONIAN SYNDROME WITH BILATERAL BABINSKI SIGN. DRS. BARRÉ and REYS.

A woman, aged 41, contracted a febrile illness followed by rapidly developing paralysis agitans. In addition, syphilis was present. The beginning of the disease antedated the epidemic encephalitis. A positive Babinski sign was the most curious feature. On both sides the putamen was almost completely degenerated. The substantia nigra also was decidedly affected. No syphilitic lesions were found in the brain. The internal capsule and pyramidal tract appeared normal. The authors believe that this case shows that syphilis is not the cause of parkinsonism.

PONTILE SYNDROME WITH LOCALIZED MYOCLONIA AND DISORDERS OF SLEEP. DR. VAN BOGAERT.

The patient in the case reported had paralysis of the limbs on the left side with involvement of the face on the right side, together with myoclonic movements of the face. There was marked somnolence. She was easily aroused, did not complain of headache or confusion and responded perfectly to questions, but as one talked to her she went to sleep. She went to sleep while eating, and would stay awake only while executing some active movement. Sleep was uninterrupted at night. Myoclonic movements were present in the muscles surrounding the mouth and eyes and involved also the platysma myoides. They were slow in rate, varying from 54 to 58 a minute, and were more easily seen when the face was relaxed with the mouth hanging open. Death was due to bronchopneumonia. Careful anatomic examination showed an extensive area of softening extending from the region of the red nucleus down close to the seventh nucleus. It involved the superior cerebellar peduncle, the outer edge of the median longitudinal fasciculus and part of the median fillet. It entirely destroyed the central ground bundle of the pons. Degenerations were traced

in this ground bundle to the capsule of the olivary nucleus. The median longitudinal fasciculus was scarcely touched. At lower levels the inferior olive and superior cerebellar peduncle showed slight changes. The author believes that the central fasciculus of the tegmentum may be a restraining tract, destruction of which allows the escape of pontobulbar centers. The disturbances of sleep suggest that a regulatory center exists close to the oculomotor centers in the immediate vicinity of the aqueduct of Sylvius.

THE MOTOR FUNCTION OF THE STRIATE BODY: A CASE OF HEMICHOREA FOLLOWED BY NECROPSY. DRS. SOUQUES and BERTRAND.

A woman, aged 54, had been healthy until the age of 4, when, during an acute febrile illness, she had developed jacksonian convulsions on the left side of the body with hemiplegia. Since the appearance of the hemiplegia, involuntary movements had been noticed on this side of the body. On examination, fifty years after the onset, there was found moderate hemichoreo-athetotic movements of the extremities, which were also somewhat underdeveloped. Death was due to infectious jaundice. Necropsy disclosed isolated cystic softening destroying the putamen almost in its entirety, as if by a punch, whereas the globus pallidus and the caudate nuclei were spared. The internal capsule was slightly involved. The host of negative clinical manifestations with similar lesions would seem to show that the age of onset is of great importance, hemichorea appearing only following lesions occurring in childhood.

CASE OF SUBACUTE ANTERIOR POLIOMYELITIS WITH NECROPSY. DRS. ALAJOUANINE, GIROT and MARTIN.

A woman, aged 40, developed rapid atrophy, weakness and ataxia in the right hand which on examination was found to be of the Aran-Duchenne type. The tendon reflexes were absent. There were no signs of cranial nerve involvement and no sensory or sphincter disorders. Spinal fluid examination gave negative results. Electrical examination showed complete reaction of degeneration in the muscles of the hand and diminished excitability in other areas. The disease progressed rapidly with circulatory and respiratory involvement, and death occurred within six months. Sections of the cord showed marked atrophy of the ventral horns in the cervical region with some perivasculitis. Attempts to reproduce the disease in rabbits and monkeys failed.

POTT'S DISEASE — PARAPLEGIA. STUDY OF THE SYMPATHETIC SYSTEM. DR. SORREL-DEJERINE.

An anatomic study was made of a case of vertebral tuberculosis resulting in destruction of the sixth, seventh and eighth thoracic vertebrae. With this there was a cold abscess in front of the rami communicantes. The cord was completely destroyed by pressure. The study of the case during life had revealed early rapid paraplegia with level signs at the eighth thoracic segment. For a while the course was benign with considerable amelioration, but later aggravation occurred. The pilomotor reflex extended as far as the inguinal region on the left and slightly below on the right. The spinal pilomotor reflex extended to above the umbilicus. A period of latency was explained by the pointing of the abscess outside the spinal canal.

CYSTICERCUS MENINGITIS. DRS. GUILLAIN, BERTRAND and PÉRON.

The clinical course in this patient was that of increased intracranial pressure without localizing signs. There was some loss of intelligence, with considerable

disturbance of memory and crises of confusion. Various tests confirmed the suspicion of increased intracranial pressure. Examination of the cerebrospinal fluid showed meningitis, neither syphilitic nor tuberculous. Necropsy revealed marked basilar meningitis with cyst formation evidently due to *Cysticercus racemosus*. In addition there were cysts within the brain, particularly in the frontal lobe and fornix. No scolices were found in the cysts.

ANEURYSM OF THE INTERNAL CAROTID SIMULATING CEREBRAL TUMOR. DRS. DEMAY, BERTRAND and PÉRISSON.

The first symptoms were mental, consisting of rapid dementia, occasional loss of consciousness and periods of confusion. The final illness began suddenly with violent headache and repeated vomiting, with marked inertia and occasional periods of agitation. Physical examination revealed mild hemiplegia with a positive Babinski sign, fine tremor of the hands and sluggish pupils. The cerebrospinal fluid was normal. The course of the disease was progressive, the hemiplegia becoming bilateral, and a diagnosis of midline tumor was made. Death occurred suddenly. Necropsy revealed a spheroid mass at the base of the brain pressing upward to the floor of the third ventricle. It was found to be an aneurysm of the right internal carotid artery.

FREEMAN, Washington, D. C.

June 3, 1926

Abst. from Rev. Neurol. **1**:1166, 1926

DIAGNOSIS AND TREATMENT OF PITUITARY TUMORS. DR. VINCENT.

An acromegalic man presented bitemporal hemianopia, headaches, vertigo and slight papilledema. In five months roentgenotherapy to the extent of about 20,000 R was given. The patient has recovered sufficiently to resume his occupation. Vincent also calls attention to the fact that the dilatation of the sella, and even the destruction of the clinoid processes seen in the roentgenogram, can be caused by other tumors of the brain, particularly those of the posterior fossae. The absence of pituitary symptoms in these cases will give the proper clue.

[ED. NOTE.—Dr. Behnken of the Physikalisch-technische Reichsanstalt, Berlin, has defined the roentgen unit (symbol "R") as follows:

"The absolute unit of the roentgen-ray dose is obtained from that roentgen-ray energy, which, by utilizing fully the secondary electrons produced, and by avoiding secondary radiations from the wall of the ionization chamber, produces in 1 cc. of atmospheric air of 18 C. (64.4 F.) and 760 mm. atmospheric pressure, such a degree of conductivity that the quantity of electricity measured by saturation current equals one electrostatic unit."

Dr. Solomon, in 1920, described an ionization unit which he called a "Roentgen" unit and designated it by the letter "R," defining it as "that amount of roentgen rays producing the same ionization as 1 Gm. of radium element at a distance of 2 cm. from the graphite ionization chamber, in the same axis after filtration through ½ mm. of platinum."

The comparison of the French with the German unit is as 2.25 is to 1. The (German) Behnken "R," therefore, is equal to 2.25 (French) Solomon "R" units, but this ratio changes with different wave lengths.]

Angle Tumor with Operation and Recovery. Drs. Vincent and Dénechau.

Two atypical symptoms in the course of the disease were marked facial paralysis and puerility. The tumor was a cholesteatoma. The patient was walking in three weeks.

Iodized Oil, 40 Per Cent, in the Inverted Position. Drs. Sicard, Haguenau and Mayer.

The iodized oil is injected by lumbar puncture and the patient inverted to 70 or 80 degrees. No disturbance such as vertigo or headache is noticed even when the oil has penetrated into the cranial cavity. When the patients resume the normal position, the oil gravitates to the sacral sac. Thus, the spinal subarachnoid space can be explored as easily as the esophagus with an opaque sound. The indications for this procedure in cases of suspected spinal subarachnoid block are obvious.

Treatment of Status Epilepticus by Injection of Phenylethylmalonylurea in the Cisterna Magna. Dr. Ayala.

Two cubic centimeters of a 10 per cent solution of phenobarbital is injected into the cisterna magna. In five patients in status epilepticus with a serious physical condition, the convulsions stopped immediately. Profound sleep resulted, lasting five or six hours, and was followed by improvement in the whole general condition.

Operative Treatment with Improvement in Two Cases of Syringomyelia. Dr. Poussepp.

The operation consisted of longitudinal incision of the dura and the arachnoid with puncture of the spinal cord by a hypodermic needle, then incision of the spinal cord in the median line. The dura was sutured and the skin closed. In both cases, within a few weeks after the operation there was a notable reduction in the extent of the loss of pain and temperature sensation. It is too early yet to say whether the improvement is permanent.

Spinal Automatic Reflex Movements in Tabetic Patients. Dr. Negro.

Automatic movements are common among ataxic tabetic patients, particularly when they are lying on the back. Painful stimulation of the sole of the foot is followed by a marked withdrawal reaction resembling a defense reflex. These movements are sometimes preceded by rapid irregular movements of the toes. voluntary movements of the patient or the sight of the legs will inhibit movements provoked by this cutaneous stimulation. A single stimulus will bring about a periodic change of such movements similar to those provoked by spinal lesion of the pyramidal tract.

Dysbasia Lordotica (Torsion Spasm) Treated with Braces. Dr. Froment.

An apparatus is described consisting of a modified sacral corset with thigh plates, adjustable with set screws. Its mobility will not permit too great flexion of the trunk on the thighs. Application of this device brought about considerable improvement in the patient's gait.

Medullary Compression After Fracture of the Sixth Cervical Vertebra in a Case of Spondylosis. Improvement Following Operation. Dr. Egaz Moniz.

Fractures in patients immobilized by rhizomelic spondylosis are fairly frequent. A porous condition of the bones favors their occurrence, but fractures

of the vertebral bodies without serious results are rare. The patient described began suffering with joint pains at the age of 25. Twenty years later he was practically rigid from the neck down. In 1924, he was thrown from a donkey and suffered pains in the cervical region, but only a year later were there signs of an organic nervous disorder consisting of weakness and pain. Examination showed reduction of pain and temperature sensibility on the right side below the eighth cervical segment with slight involvement of the left side, together with Horner's syndrome. The roentgenogram showed fusion of the vertebrae and fracture of the body of the sixth cervical vertebra. There was no spinal subarachnoid block. The immobilization of the spine was favorable for keeping the head in position, and the operation consisted of excision of the spines and laminae of the fifth, sixth and seventh cervical vertebrae. There were dural cicatrices but no medullary compression. The subjective and objective sensory disturbances were moderately benefited.

THE SPINAL CORD OF YOUNG RABBITS BORN OF FEMALES INTOXICATED BY NICOTINE. DR. CATOLA.

Myelinization of the spinal cord at birth is less developed in such rabbits than in normal individuals, particularly in the inferior thoracic and lumbar regions.

FURTHER OBSERVATION OF A PULSATING SACRAL SARCOMA AFTER ROENTGENOTHERAPY. DR. BÉCLÈRE.

When this patient was previously shown the result was considered successful, but recurrence was feared. This prophecy was fulfilled, for a nonpulsating mass recurred and numerous metastases developed which resulted in death.

DANGERS TO BE AVOIDED IN ROENTGENOTHERAPY OF INTRACRANIAL AND INTRA-SPINAL TUMORS. DR. BÉCLÈRE.

Roentgenotherapy of diseases of the central nervous system dates from 1902, when Raymond noted its efficacy in a case of syringomyelia. In 1909, Béclère, using a new technic, attacked tumors of the hypophysis. Only in 1915 was the treatment applied to tumors of the cerebrum and cerebellum by Nordentoft. Results are extremely variable depending on the unequal sensibility of various cellular elements, normal and pathologic, and on the rapid reduction in the lethal powers of the rays on deep penetration. Formerly only radiodermatitis was to be avoided, but now under more powerful and better controlled dosage radionecrotic accidents as well as mechanical and toxic ones are to be found. Mechanical and toxic accidents are to be feared. Mechanical accidents are probably the most serious in dealing with the craniospinal cavity. Similar accidents are found in cases of incarcerated tumors located anywhere.

The phenomena seem to depend on the inflammatory edema produced in the irradiated growth. They appear within twenty-four hours after the first irradiation, and symptoms differ according to the site of the lesion. In cases of spinal neoplasms, the spontaneous pains are exaggerated and become radicular in distribution. Sphincters become incompetent, and acute decubitus, fever and cutaneous blebs appear, similar to those observed in traumatic section of the spinal cord. In other words, the medullary compression is temporarily increased by the irradiation. The intensity of these accidents varies less with the radiosensibility of the tumors than with the dosage and the degree of medullary compression already present. Heavy doses of roentgen rays are therefore dangerous in severe medullary compression whether in the interior of the

cranial cavity or in the spinal canal. Hyperemia and edema quickly following roentgenotherapy are dangerous and may even soon cause death, according to the observations of Fischer. The accidents are explainable by the intracranial hypertension and its results as shown by measurements of pressure of the cerebrospinal fluid. It would be well to begin treatment by low doses of roentgen rays, particularly if the spinal fluid pressure is increased. Intrasellar tumors have been attacked with probably greater success than the others, although accidents are occasionally observed in dealing with them. With an intracranial tumor to treat, one is faced by the dilemma of too severe a reaction from overdosage and insufficient killing power on the tumor cells from feeble doses. Béclère believes that roentgenotherapy is often excellent treatment and capable of giving good results, but his motto is "primum non nocere."

Rare Case of Medullary Compression (Projectile Latent for Twenty-Six Years). Dr. Porot.

The patient, aged 50, developed slowly progressive paraplegia. In 1900, he was wounded in the chest 3 cm. above the left nipple, but recovered after two or three weeks. There was no weakness of the limbs. In 1926 he suddenly developed pains in the back and noticed that his legs dragged. There was weakness of the limbs and some ataxia. The tendon reflexes were practically normal. The Babinski sign was indefinite. The legs were hypertonic. There was a zone of anesthesia below the umbilicus with hyperesthesia above. The roentgenogram showed an irregular mass opposite the ninth and tenth vertebrae. Iodized oil, 40 per cent, was stopped completely at this level. At operation a calcareous mass was found adherent to the vertebral bodies and laminae on the left side; it was of stony hardness and contained many fragments of the bullet. There was some hyperemia with adhesions of the meninges, and the cord was somewhat compressed. Operation was followed by death.

Posterior Rhizotomy for Causalgia of the Upper Limb—A Study of the Sensory and Motor Disorders After Operation. Drs. Sicard, Hagueneau and Mayer.

The patient was wounded in the right wrist in 1915. Several operations were performed for the extraction of small foreign bodies, but the patient continued to suffer from crises of severe pain which interfered with sleep and were not relieved by brachial sympathectomy. The dorsal roots were cut from the fifth cervical to the third thoracic. The patient has not suffered since the operation. The arm feels heavy but not painful. He loses it in bed and must look at it in order to grasp it. He forgets it completely and sometimes unconsciously the arm makes a sudden violent synkinetic movement which surprises him. There is complete loss of the sense of position and weight. Bony sensibility is abolished. Passive movements are perfect. Voluntary movements have lost nothing in force; incoordination is striking, however. The hand or finger travels suddenly off in the wrong direction. The goal is passed, the hand is brought back by a number of clumsy movements recalling tabes or multiple sclerosis. The ataxia is even more marked when the eyes are closed. The tendon reflexes are abolished. The local pilomotor reflex is marked. There are no vasomotor or trophic disorders, but slight myosis is present on the side operated on. The operation seems to have been worth while because the pain has disappeared.

Freeman, Washington, D. C.

Book Reviews

TWENTY YEAR'S EXPERIENCE OF OBJECTIVE STUDY OF THE HIGHEST NERVOUS ACTIVITY OF ANIMALS. By I. P. PAWLOW. Price, 3 rubles (about $1.75). Leningrad-Moscow: Government Publication, 1925, pp. 397.

THE METHODS OF RESEARCH IN CONDITIONED REFLEXES. By N. A. PODKOPAEV. Price, 1 ruble (about $0.60). Government Publication, 1926, pp. 64.

"The objective method of research is the only one that will gradually open up the full analysis of that vast and limitless adaptation which comprises life in all its scope. Are not the strivings of the plant to light and the search for truth by mathematical analysis phenomena of one and the same type essentially? Are they not the last links in an almost endless chain of adaptation met with throughout the whole living world? We can analyze adaptation on an objective basis in the simple forms; why then change our method when we study forms of the highest type? . . . The objective study of living substance beginning with the study of tropisms in the elementary types of organisms can and must remain such even when we reach the most complex, so-called psychic, phenomena of the highest animals."

Thus the first contribution to "Twenty Years' Experience" — the contribution with which Pawlow first gave the international world his "new method" in a speech at Madrid, in 1903 — is introduced, and this idea pervades all his work. Like Galileo, the creator of experiment, he too uses the true inductive method.

For the Aristotelean biologist, life in its various forms is only a continuous chain of gradations. The differences between types are much more of degree than of kind; the passage from one into the other is ever imminent and at no stage is he compelled to transcend. "Fundamentally, the strivings of the plant to light and the search of the mathematician for truth are phenomena of the same type," and so the physiologist can start anywhere in the chain even if his object is to find the laws and system of human behavior, or rather behavior in general. For the physiologist, Pawlow argues, behavior of the highest animals should remain the subject of physiologic research; that is, should be definable in terms of function of one of the organs of that animal. In studying function everywhere else, the physiologist remains on a strictly objective observing platform simply investigating reaction to stimulus. Then, why should we change our method of approach when we study behavior? From the biologic point of view, behavior is fundamentally a matter of adaptation of the animal to its environment and the constant changes therein, and if physiology is to attempt at all to study this function, all of it should be approached from the same point of view. All adaptation, all behavior, must have the same fundamental laws, or none of it can come within the reach of the physiologist.

Pawlow is studying the function of salivary glands in the dog. He is at once confronted with the first difficulties in the objective study of behavior — adaptability — for phenomena encountered here seem to break up into two distinct types. When the dog is given hard, dry food, its salivary glands secrete a great deal of limpid saliva rich in mucus, which helps the mastication and deglutition of such food. Moist and soft food, on the other hand, that is easier to masticate and swallow, calls forth a secretion of much less saliva and one of a different type. Again, when dry sand is placed in the dog's mouth,

there is a copious flow of watery saliva which contains hardly any mucin at all — the sand is to be thrown out of the mouth, and all that is needed is enough fluid to wash the mouth clean; when small, clean pebbles are placed in the mouth, they call forth no secretion of saliva at all, for there is no need of washing the mucous membrane. Here, then, we find adaptation of the dog to its environment and the changes in it, but this seems to be a process in which a physiologist can remain on his objective, scientific platform. Long before the time of Pawlow the idea of the reflex arc was introduced into physiology and, so far, adaptation can be understood in terms of reflex function. The type of food introduced and its physicochemical properties stimulate centripetal nerves; these stimulate the center and another set of centrifugal nerves determines the type of secretion necessary.

It is necessary to go only one step further to find an apparently different type of reaction. When the dog is shown a certain type of food, it may react with an appropriate type of saliva. Even such things as the sight of the man who feeds it its daily portion, the sound of his voice, and various other apparently indifferent stimuli may call forth perfectly appropriate reactions. These were known as psychic reactions. We used to speak of likes and dislikes, desires and even such things as thought and memory in the dog as the foundation of these reactions. The zoopsychologist would speak of an internal subjective experience of the animal and would attempt to systematize these experiences by comparing them with the various experiences that we have on such occasions. If this were true, and if psychology should concede and take this as the appropriate point of view, it would have to give up its objective attitude. This would not be so bad, but where are the evidences that these guesses at wishes, desires, memory and thought in an animal, which one can only observe but in no other way consciously communicate with, are facts? There are no such evidences, and even if it is assumed that the guesses are correct, no definite clearcut scientific formulations could be founded on them.

Physiology, if it is to remain a natural science, will have to remain on an objective platform, and in these so-called psychic phenomena the same laws should be found as in the others that are physiologically approachable; Pawlow approaches this field on the same objective basis. The first type of adaptation bears a purely reflex nature and is rigid, permanent and constant. The stimuli that call forth such reflexes must come into direct contact with the nerves of the organ that reacts to them. Such a reflex we can call permanent or nonconditioned. The other reaction seems to be capricious, does not depend on actual contact with the organ in question and seems to be temporary. What objectively definable laws can we see there? One feature was evident from the beginning. The "indifferent" stimuli that would call forth such a reaction had to coincide at some time or another with some one of the permanent nonconditioned stimuli. With this in view, Pawlow set out to inquire and formulate the laws that govern such reactions, if such laws there be. The dog is given a certain type of food, and every time this food is given, an indifferent stimulus, say the vibration of a tuning fork of a certain frequency, is brought into play. After a few repetitions, the tuning fork is itself sufficient to call forth a secretion of saliva and of a type that is appropriate for the type of food that served as the first nonconditioned stimulus. Thus a new reflex was established, but a reflex by virtue not of the value of its own stimulus but of that of the nonconditioned one. This, then, Pawlow calls a conditioned reflex and the stimulus a conditioned stimulus, and he defines it as a temporary association between environmental phenomena and reactions of the animal to them.

The animal at any given moment in an environment is subject to constant flow of change. It brings with it into this constantly changing milieu a certain number of inborn, rigidly laid down, nonconditioned forms of reaction. This, the foundation of all its behavior, actually comprises a small sector of the rich system of reactions which it presents to the observer. The greatest part of its behavior consists of these new associations of acquired, temporary "conditioned" reflexes. The most important property of the conditioned reflex is its temporariness. As already mentioned, the fundamental condition for the development of such a reflex is coincidence with an unconditioned reflex. Given such coincidence, it can afterward by itself call forth an appropriate reaction. This, however, is not constant. If the conditioned reflex is repeated at short intervals (two minutes) without the nonconditioned stimulus, the reaction will gradually diminish and then be rendered "extinct." The biologic value is obvious. It is just this temporariness that makes the conditioned reflex valuable as a signal of the nearness of food or the approach of an enemy. The extinction of the conditioned reflex, however, as will be seen later, is temporary too and is only a form of inhibition.

Thus the ability to form temporary reflexes serves to synthesize different elements in the environment into biologic wholes, but the elements must be picked. If the signal is to be represented by vibrations of a frequency of 1,000, it will not do to confuse other frequencies with it. Actually a new mechanism springs into light in the form of analyzers. The vibrations of a tuning fork of a frequency of 1,000 conditioned by a certain type of food will, at first, be confounded with other sounds within certain limits; but on repeating this vibration frequency with the nonconditioned reflex, and especially by repeating other sounds and other frequencies without the nonconditioned stimulus, we can gradually bring the limit range of the stimuli that will call forth the reflex down to astounding narrowness. The dog will thus differentiate 1,000 from 1,012 vibrations and certainly from all other kinds of acoustic stimuli. This function of analysis or differentiation is again dependent on a certain form of inhibition.

Physiologically, the analyzer can be looked on as a nerve mechanism consisting of the following parts: (1) a certain peripheral end-organ (eye, ear, etc.); (2) a corresponding nerve, and (3) the central end of this nerve, i. e., the group of cells in the cerebral cortex in which this nerve ends (physiologically). The part we are interested in here is the third, and Pawlow believes, and has proofs to that effect, that it is here that the final analysis of the external world takes place. This analysis takes place gradually, as was outlined before, is entirely dependent on the mechanisms of excitation, irradiation, inhibition and concentration and is subject to the special mechanism of release. These may be considered in detail:

"There is one condition, the presence or absence of which will decide whether the impulse brought from the outside into the cerebral hemispheres will call forth a process of excitation or inhibition for any length of time (in other words, will be positive or negative). If this stimulus coincides with any other extensive stimulation in the hemispheres, or lower cranial centers, it will remain positive. If it does not, it will sooner or later begin to act negatively. Both these processes have the property of spreading to other points and if in so doing a positive one, coming from the one point, crosses the path of a negative one, coming from another point, a conflict will ensue, the result of which will be a reciprocal limitation to definite regions (these, of course, physiologically and not anatomically speaking); thus, there is an ever present

strife, the balancing of which gives us normal behavior." In this way, Pawlow sums up his views on all these complicated mechanisms. First let us consider that of inhibition, as we have already discussed that of excitation.

Inhibitory processes fall into two large groups:

1. *External Inhibition.*—This represents the mechanism of the so-called Wettstreit, a well-known physiologic mechanism. It means the successful combat of an external, indifferent stimulus carried to the field of activity of a reflex in action. Thus, the flow of saliva caused by the sound of a bell, conditioned by its coincidence with the administration of some type of food, will cease or will be materially diminished if the sound of a toy trumpet happens to be brought into play.

2. *Internal Inhibition.*—This has its foundation in the peculiar relationship that exists between the new reflex and the old one by the aid of which it was conditioned. There are four types: (*a*) Extinction: If the conditioned stimulus is repeated several times at short intervals (two minutes) without the nonconditioned one, its effect will gradually weaken and then disappear. (*b*) Delayed Reflex: The conditioned reflex is formed by the process of having the nonconditioned follow the conditioned stimulus almost at once (from three to five seconds). If, after establishing such a reflex, we repeat the process several times but allow an interval of three minutes to elapse between the conditioned and the nonconditioned stimuli and then try the conditioned stimulus alone, we will find a delay of from one and one-half to two minutes between the beginning of the conditioned stimulus and the flow of saliva. (*c*) The Differentiation Inhibition: The perfection of the analyzing process is brought about by a temporary inhibition of all other points except the one that corresponds to the particular stimulus. This will be further elaborated in the description of concentration. (*d*) Conditioned Inhibition: The continuous repetition of a conditioned stimulus accompanied simultaneously by an indifferent one and not followed by the nonconditioned one, will render the indifferent stimulus an inhibitory agent if we repeat the same conditioned stimulus by itself reinforced by the nonconditioned one. Let us suppose that the sound of a bell was conditioned by the administration of meat. The sound of the bell will, after a while, cause a flow of a certain type of saliva. If we now bring into play the effect of a flashlight simultaneously with the ringing of a bell, but without the administration of meat, and repeat this several times while we alternate these with the sound of a bell followed by the administration of meat, the flashlight will become a conditioned inhibitor. Moreover, the effect of both the flashlight and the bell will also be inhibitory.

The mechanism of internal inhibition, like that of the conditioned reflex, is valuable in the process of adaptation because of its temporariness. Like the conditioned reflex, internal inhibition is liable to inhibition in its turn, as an internal inhibitor is always, in a broad sense, a conditioned inhibitor, and the mechanism that it inhibits can be set free. This is the mechanism of release (disinhibition). Thus, when some new and indifferent stimulus, e. g., the sound of the gramophone, comes into play during the action of a conditioned inhibitor (flashlight plus bell) a flow of saliva occurs. In other words, the conditioned reflex was released. This occurs similarly in other types. Here again, then, we see a guard against a too rigid adaptation to some certain type of environment.

Irradiation and Concentration.—The mechanism of these two processes can be best illustrated by the following example: Five distinct points are taken on the skin of the dog's leg at equal distances from one another, the first at the

shoulder and the last at the toes. The four upper ones are conditioned; that is, a certain tactile stimulation of these is followed by a certain type of food. A similar stimulation of the fifth point is repeated without food. Now, when we stimulate each of these points at long enough intervals, we get a flow of saliva in the case of the upper four, and none in the fifth. The fifth point is now stimulated without effect on the salivary gland. After a short interval, the one nearest to it is stimulated, and there is no flow of saliva, whereas with an interval of shorter duration the excitation of the second nearest gives a diminished flow of saliva; in the third the diminution is less, and the fourth gives the normal amount of saliva. By varying the length of intervals, we find a specific time for each one of these points. The negative effect of the fifth point, i. e., the absence of flow of saliva, was brought about by an internal inhibition, owing to the repetition of the stimulation of it without the administration of food (this is also shown by the fact that before it became a negative factor it caused a flow of saliva, and only on repeated stimulation without food did it take on its negative properties). When this inhibitory activity is excited, it spreads at a certain speed to the other points, inhibiting them too. This is the mechanism of irradiation.

If the interval of time between the excitation of the fifth point and the one nearest to it is longer than was necessary to cause complete absence of flow of saliva on exciting the last one, we begin to have a gradual return to normal. This occurs similarly with the points further away, but the intervals of time have different proportions. This return to normal constitutes the mechanism of concentration. Both of these mechanisms are also found in the process of excitation. They may be expressed in the following law: "Any exciting or inhibiting process, started at some point in the cerebral hemispheres by virtue of definite peripheral stimulants, is subject to certain types of movements along the surface of the hemispheres, the rate of which can be measured not only by seconds, but also by minutes. These movements are in two directions: (1) spreading (irradiation); (2) return to original point (concentration)."

These are, according to Pawlow, the fundamentals of the function of the highest centers of the nervous system. With the keen insight that characterizes all these fundamental studies, Pawlow takes great care not to confound functional with structural centers. When he speaks of centers of food, of defense, of freedom and the like, he is careful not to fetter these to special, localizable and clearcut anatomic centers. As a physiologist, however, he cannot remain satisfied with mere names that would stand for things intangible. These centers, the sites of his mechanisms, he insists are in the cerebral cortex even though they do overlap one another. With this in mind, he undertook numerous experiments with extirpations of various parts of the cortex in his laboratories. and he came to the conclusion that each and every analyzer possesses as distinct a cerebral center as it does a peripheral end-organ. In fact, the whole cerebral cortex consists of these centers of the analyzers, and nothing else but them. This would mean, and Pawlow makes it quite clear, that the entire cerebral cortex is receptor and contains no effector parts. A great deal of work was done to prove that the gyrus centralis anterior, the so-called motor area, is also receptor, but is receptor of one of what he calls the internal analyzers — the analyzer of motility. The idea that some parts of the cortex are sovereign to others, e. g., that the frontal lobes are the superimposed psychic structures that in some way control or influence all other sensory and motor organs, is done away with. A complete democracy and equality of function are established all over the cortex.

All his work in this direction, which is extremely fascinating, is, however, too complicated and as yet too unfinished to be considered here. One factor, however, should be considered now. Each one of the centers represents the point where impressions received in the peripheral end-organ are analyzed and then synthesized into conditioned reflexes. Injury to or disease of such a center will interfere with both these functions, depending on its extent. So injury to the occipital lobe will cause what used to be called psychic blindness. In a case of this type the dog will be said to "see" but not "recognize." It will avoid obstacles in walking, but will not recognize its master, for instance. Previously, as Pawlow puts it, the zoopsychologist spoke of the dog seeing but not understanding. Pawlow sees in this condition only a decrease in the power of the analyzers. The coarser the analysis required, the lesser its vulnerability in brain injury. So Pawlow succeeded in showing that partial removal of the occipital lobes brought about a condition wherein the dog could still differentiate light from darkness and form conditioned reflexes dependent on this differentiation, but more complicated visual conditioned reflexes could not be formed. Different gradations of injuries to various centers resulted in corresponding gradations of diminution in the delicacy of the respective analyzers.

The work started on these relatively simply formulated mechanisms is gradually spreading to include and systematize the various activities and phenomena of animal behavior. An interesting series of experiments is conducted on the problem of sleep. According to Pawlow, sleep is only a further elaboration of internal inhibition. Internal inhibition is gradually being formulated to mean partial localized sleep, and sleep thus to mean an irradiation and spreading of internal inhibition over the whole hemispheres and even the midbrain. It appears that when a delayed "reflex" is drawn out over a long period of time the animal will fall asleep. Pawlow believes that "any more or less prolonged stimulation of some one definite point of the cerebral hemispheres, no matter how important or unimportant or how strong, if it be unaccompanied by stimulations at other points of the hemispheres, or not replaced by other stimulants, it will sooner or later, without fail, lead to sleepiness and sleep."

With the study of the normal function of the cerebral cortex (normal behavior) Pawlow has recently begun to investigate abnormal functions. In his preface to this, the third edition, he says: "The central factor in the activity of the cerebral hemispheres is the relation between the exciting and inhibiting processes. The balance between these two, and the deviations from it within normal limits and outside of them, determines all our behavior in health and disease." In his experiments in which he attempts to determine the ability of the animal to check exciting processes by inhibiting ones, and vice versa, he comes to the conclusion that the dog can only go to certain limits and no further. Outside these, the delicate balance is overturned and the animal loses its ability to analyze and concentrate properly, and goes off either to one extreme or the other. We get the following abnormal condition: (1) Overexcitation and hypertonicity, when inhibition becomes altogether impossible or very difficult, and thus only coarse differences can be analyzed. (2) Predominance of inhibition in the following forms: (*a*) when all stimuli, independent of their intensity, act equally; (*b*) paradox, when only weak stimuli produce inhibition; (*c*) ultraparadox, when previously developed inhibitors act as excitors.

These conditions, which Pawlow regards as experimental neuroses, seem to develop when the demands on the adaptability of the animal, i. e., its ability to balance exciting and inhibitory processes, are particularly strenuous. The condition that brings them about generally takes the form of a constellation of circumstances rather than of any single factor. One experiment is particularly interesting. Two dogs, one of a very active and the other of a phlegmatic, quiet disposition, are chosen. A difficult problem, that of analytic differentiation of delayed reflex (three minutes), is presented to both. Both have difficulties with it, but after a while again respond to it without any abnormal manifestations. Then this problem is further complicated in the following fashion: The nonconditioned stimulus (food) is left as before, but for the previously indifferent conditioned stimulus, a painful one (electric current), is substituted. The use of painful or, as Pawlow calls them, destructive agents, as conditioned stimuli, brings in even under ordinary circumstances a difficult problem. The dog has to overcome its tendency to react with defensive measures, inhibit these and react to the painful stimulus as if it were a pleasure-bearing one (because it is conditioned to represent food). In this way the difficulties were increased, and after the formation of the delayed reflex by conditioning the pain-bearing stimuli, a few repetitions were sufficient to throw both dogs into a markedly abnormal state. The active dog lost altogether, or to a certain extent, all of its acquired, inhibitory reactions, whereas in the phlegmatic dog this happened to the acquired, exciting (positive) reflexes. In both, this pathologic condition remained unchanged for a long while, and only the administration of bromides helped to bring the first dog back to normal; all efforts to "cure" the phlegmatic dog were of no avail.

The evaluation of all these experiments, as can be surmised, is a difficult task. When one creates an "environment" for the animal with a view of analyzing behavior as conditioned by various factors in this environment, one has to be master of the situation. Elements that may affect the animal and yet escape the attention of the observer or be outside his control will, of course, make the results much less reliable. Difficulties have been encountered, obstacles overcome, a wealth of material in the form of facts has been noted, and Pawlow has gradually built up a method and constructed a laboratory and technic which come as near perfection as seems at all possible. N. A. Podkopaev, in his short monograph, gives a concise but faithful account of these. The operation for a permanent fistula, the construction of special laboratories and apparatus, the technic of conditioned reflexes, inhibitions, releases, etc., with the sources of error down even to the special care of the animals, are clearly and fully described. Finally, both Pawlow and Podkopaev give a full bibliography of the literature on the subject which, it is to be regretted, is almost exclusively in the Russian language. One might add here that G. V. Anrep is now translating a series of lectures on the subject by Pawlow; he expects to have these published in English within the next few months.

Ever and again, as long as man will search life for the illusive unknown, as long as man will think, Plato and Aristotle under different forms but with the same bend of mind will recur. Of the philosophy in Pawlow's work, the idealist will say with Descartes, "Galileo thinks better than the others in that he thinks mathematically, but he lacks a real foundation because he looks for special effects of nature, but not for the final fundamentals of it." In fact, both search for fundamental principles, but the one seems to be concerned with the "why" and the other with the "how."

This is no place to discuss Pawlow's philosophy; one point, however, should be considered. Pawlow's turn to the purely objective method in his study of the behavior of dogs is one, the justification of which no philosopher will doubt. It is a purely Kantian turn. What the dog feels, what he experiences subjectively, we, as rational beings, have no means to know; we can only observe, and being scientists, should not guess. Science should always remain within rational bounds, within the limits of logic. But Pawlow is not concerned with the behavior of dogs only. His physiology aims to include "the knowledge of the mechanisms and the laws of human nature . . . to reach a true, a complete and a lasting human happiness." Here, however, we must say that ratio is only one of the functions of the human psyche and logic its peculiar weapon whereby it both functions and limits itself. Ratio, logic and categories are peculiar to consciousness. What of the irrational, the illogical and antilogical, what of feelings and intuition? What of a deeper and higher understanding that lies beyond the grasp of ratio (dianoia), the field of the Platonic noesis? But ever and again, these questions will recur, and there will always be those for whom the Protagorian maxim "man is the measure of all things," the all-inclusiveness of science, will be sufficient.

It is in the field of science, however, in that of exact neurophysiology, and to a certain extent of psychology of the objective, experimental type, that the value of Pawlow's work is of the greatest importance. No longer does physiology have to deal with intangible, imaginary contents. Behavior from this point of view can and must be made the object of exact study. Its value will especially be appreciated by the clinical neurologist. It bids fair to help to bridge that gap that existed between the attempt at localization of psychic functions and their understanding. The whole field of aphasias, agnosias and of all the psychic representations of special sense disturbances will be better understood. The time can also be looked forward to when this work will help to throw the light of exact evaluation on a great many aspects of psychiatry that are as yet covered up in the twilight of nosologic, empiric description.

"Twenty Years' Experience" looked at from any point of view is worthy of becoming a classic study in the field of biologic endeavor. The method of compilation of the different essays, lectures, etc., in chronologic sequence, which gives one the history of the development yet without the disadvantage of retrospective coloring, makes the study of it particularly fascinating. It tells the story of years of scientific striving with all the waverings, fears and hopes, possible failures and successes, but always with the same query in the background, the same goal ahead. The presentation is clear and mathematically precise, and the critical attitude of the scientist is preserved even where the heights scaled bespeak the attempts at sounding the very mysteries of human nature.

OUTLINE OF ABNORMAL PSYCHOLOGY. By WILLIAM MCDOUGALL. Price, $4.00. Pp. 566. New York: Charles Scribner's Sons, 1926.

The attitude of the reader as to the value of this book will necessarily be the same as that toward the first volume, the "Outlines of Psychology." If he found that work valuable, so too he will find this one. In that volume, the author has practically reached the maturity of his theoretical thinking. In this volume, he resorts to the same theoretical foundations and the same methods of attack. The material differs; that is all. He has simply gone somewhat beyond the normal, yet not far enough to consider all the problems of abnormal psychology. He limits himself to disordered processes that seem to him of a

purely functional nature. "I hope," he states, "to bridge the gap between academic psychology and the study of the neuroses and psychoses, a gap which remains regrettably wide." The persistence of this gap, which certainly needs filling in, he explains as follows: "The breach I seek to heal has arisen largely from the fact that academic psychologists, for the most part, have enjoyed neither medical training nor extensive opportunities for the study of mental and nervous disorders, while the medical men who have devoted themselves to such studies have had as a rule little training in or contact with academic psychology, a state of affairs well-nigh inevitable and not easily to be remedied in the future." He considers this volume as "an early, if not the first, attempt to bring into something like systematic order a very large and difficult province of science, hitherto cultivated in patches rather than as a whole."

The author proceeds to present his credentials for this monumental task, referring to his interest and training in academic psychology, his medical training and his close contact with nervous and mental cases, particularly during the World War.

The writer claims membership in a school of abnormal psychology "that recognizes human nature as founded on instinctive tendencies, and as everywhere always manifesting purposive striving, rooted in the instincts." This purposive striving manifests itself "on very different planes of consciousness; that besides our most fully conscious purposes, our organs express and are controlled by purposes of which we may be only very obscurely aware, and also by purposes which operate wholly on a subconscious plane." This theory also offers a basis for differentiating higher from lower forms of mental activity. To quote: "All mental life is purposive, and that the higher forms of mental activity differ from the lower, not that purpose is added to them but rather that in the higher forms the goal is clearly defined in consciousness while in the lower forms it is obscure and vague." This theory he also applies to the abnormal, stating that "functional disorder is, in a sense, always purposive, an expression, however distorted and imperfect, of purposes however obscure and unacknowledged."

Throughout, his hormic theory of instincts, hormic in that "each instinct seems to be in some sense a great reservoir of energy, or at least something that liberates energy and directs it toward the instinctive goal proper to the species," is much used for purposes of explanation. He takes occasion to express absolute conviction that his hormic theory of human and animal nature expounded many years ago in his "Social Psychology" is correct. His attitude toward this question becomes apparent in the following lines: "Since writing that book, further reflection and more intensive study of the phenomena of mental disorder have convinced me that the theory is along right lines, that all other types of psychology are of little value." And then we journey into his emotional reactions to its reception by critics, which unfold themselves in these words: "And in spite of the many attacks made on it, more especially in America, of recent years by dogmatic young mechanists, I have been confirmed in my conviction by finding that it is in line with fundamental, though often merely implicit, assumptions of all the most progressive and fruitful workers in the field of psychopathology, of Freud and Jung and Adler and Stekel, and their many followers, of Morton Prince, Rivers, Bernard Hart, William Brown, Crichton Miller and of many others of that eclectic school with which I affiliate myself."

After a brief sketch of the various schools of abnormal psychology, he proceeds to the second chapter in which the nature of functional disorders is

discussed. The close student of this field is already familiar with most of the material, which appeared in an address made to the American Psychiatric Association in 1921.

The third chapter considers fatigue, drugs and sleep. Most attention is given to drugs, and the material on alcohol is from a chapter in a small work published during the war by the British Liquor Control Board.

Then fellow chapters on hypnosis. They, perhaps, are the best in the book, manifesting much experience and painstaking theoretical study. Here again the student of abnormal psychology is familiar with this attitude; the author cites at considerable length an article published in *Brain*, in 1908, entitled "The State of the Brain During Hypnosis." He elaborates by presenting a theory of suggestion which maintains that "the human species is endowed with the instinct of submission; and that, with the development of language and intellect, verbal indications of the attitudes of the strong become very important means of working and directing this submissive impulse; that the impulse, the emotional conative tendency of this instinct, is the main conative factor at work in all instances of true suggestion, whether waking or hypnotic." Finally, he considers "rapport, the essence of which is that the power of suggestion — is much exaggerated." The rapport, he states, "between the operator and the hypnotic subject is essentially the relation of prestige and submission." He explains why suggestions have such great power on the brain that the state of relative dissociation obtaining during hypnosis hampers all intellectual operations by means of which the suggestions received might be criticized, and that the operator is always playing on and keeping in action the submissive instinct of the subject; this instinct, according to the depth of hypnosis, becomes a secondary split-off personality, its impulse being wholly directed to the operator.

The section on dreams also involves much thought and some original material of a valuable character. The dream interpretations according to the methods of Freud and of Jung leave much to be desired, as the author himself points out. In the chapter on day dreaming, autistic thinking does not receive much constructive consideration but its discussion gives opportunity to attack the inadequacy of the older academic psychology which, according to McDougall, has only one active principle, "that of association and associative reproduction"; he speaks of "that banal psychology which lends itself so readily to translation into terms of mechanism and heredity" and adds that "so long as psychiatry based itself on psychology of that sort, it remained ineffective and unprogressive, incapable of achieving any insight into mental disorders."

He is far more indulgent to the method of psychanalysis. Concerning freudian theories, he states that "some features are approximations to truth, especially the psychogenic nature of functional disorders, the importance of internal conflict and repression, and of subconscious working of repressed conations." And above all it is admitted by the writer "that the fundamental notion guiding Freud's therapy is correct; namely, the notion that exploration of the mental nature and origins of the disorder and the revelation of them to the patient is the most essential step in therapy." We are consequently not surprised to find much space devoted to repression and conflict. Dissociation also receives much attention, and he elaborates on its differentiation from repression. The complex also receives much consideration and he gives a neural scheme of the complex.

Delusions and hallucinations are considered theoretically, with discussion of the validity of Prince's theory of hallucination; namely, that it is a partial reflection in the primary consciousness of a subconscious stream of thinking. Symbolic symptoms also receive considerable attention.

These conceptions lead to the chapters on schizophrenia, manic-depressive disorders, psychotherapeutic methods and alternating personalities, all of which are handled in a most interesting manner. In the chapters on the theory of personality, integration and disintegration, the theoretical construction of the writer reaches its climax.

PSYCHOLOGIC STUDIES ON THE OLFACTORY SENSE. By HANS HENNING. Volume A, part 4, in Abderhalden's "Handbuch der biologischen Arbeitsmethoden." Price, $4.50. Pp. 741-836. Berlin and Vienna: Urban & Schwarzenberg. 1926.

Most of the difficulties and sources of error encountered by the experimental investigator of the special sense psychology are attributed by the advocate of strictly objective investigation to the subjective coloring that both the investigator and the individual tested bring in. There is no doubt that this coloring can be a source of error; but if man is to study the psychology of his like, how is he to avoid it? The only way out seems to be the recognition of this factor and its proper consideration in evaluating experiments. In the present monograph, the author shows how one can, in such fashion, approach a strictly scientific investigation of the olfactory sense. With the thoroughness so typical of his numerous studies in the field of psychology, he presents all phases of the subject in a comparatively small monograph, yet contriving to preserve simplicity as well as clearness.

The work is introduced by some general considerations that are to be observed in the exposition of the stimulus. We are dealing with the determination of the effects (in the form of perception) of an odorus substance dissolved in air on the olfactory sense. We will consequently have to assure physical and physiologic conditions that will be as constant as possible. The exposition may be unilateral or bilateral, equal on both sides or unequal; the person tested may be informed or uninformed as to what is done. The effects of practice and fatigue, localization of the stimulant in the organ itself or externally and the pleasantness or unpleasantness of the sensation are all to be considered before one proceeds to record values. The numerous apparatus described are constructed in the form of different devices by which one can determine the amount of the stimulant in question (by weight or volume) and its distance from the perceiving organ that are necessary to make it just perceptible.

In the determination of such values we will, of course, first have to consider the physical laws under which the stimulation takes place. Thus we will first have to determine the coefficient of diffusion, pressure in the atmosphere and evaporation of the stimulant in question, its adsorption to the walls of the apparatus used, and the effects of temperature, etc., on all these. With this determination in mind we can proceed to the study of the psychologic values; that is, the stimulus threshold, difference threshold, and reaction times, as well as the fluctuations of these brought about by altering conditions. The quality of the odor will change with the change of intensity of the stimulus, with the anticipation of the person tested, and with the residual effects of the odors preceding the one in question. The last named condition (residual effects) deserves special consideration because of its complexity. Every individual is

always under the effect of not only one or a few residuals but also of a complex "structural residua-system." The odor in question will be related to this system — first, by the memories or interrelations that its name suggests and second, by its effects on other senses (e. g., taste) which it may stimulate now or may have stimulated before.

This leads to the consideration of the general psychic phenomena called into play by the odoriferous stimulations and the relation of the odor perception to other psychologic functions. Each organ of special sense contains centrifugal as well as centripetal fibers. Besides this, as the center of this organ is related to other special sense centers in particular as well as to the function of the central nervous system in general, the centrifugal fibers may bring influences of general psychic value to bear on the apparently purely peripheral part of the function. The first of these influences is that of memory. In the olfactory sense we do not meet with the negative after-effects that are the usual thing in the optic sense, but we have positive ones similar to the "eidetic" experiences described by Jaensch in the optic sense. They are, however, much more effective and striking here. Then, as in other senses, we have the effects of associations of an odor not only with other odors but with functions of other sense organs (taste, sight, etc.), real as well as imaginary.

The next factor to consider is the influence of the emotional components brought into play. The relations of the odor in question to the whole life of the person, his milieu, class, religion, customs and sex may change not only the threshold and reaction time values but even the very quality of the odor. One must bear in mind that an odor perception, just as a perception of any other kind, cannot be considered as an individual, sharply outlined function, but as a whole psychic experience. Thus, every perception receives a so-called "Gestalt" (form) quality within which the particular special sense component (here olfactory) may be measured but not entirely isolated. The other components will always exist in the whole and will affect any single one to a great extent.

Whether the source of stimulation is physicochemically an element or compound, the perception of it as one or the other will again depend on the psychologic state and constellation of factors in the individual. Any combination of stimuli can thus "melt" together into one and appear as such to him. This melting may be a more or less permanent one, or one that breaks up immediately after the first impression and admits of subsequent analysis into the different components.

Outside of these psychologic influences, one has to consider those of a more objectively physiologic type. Partial or total anosmia, hyposmia or hyperosmia, of course have to be considered before one is to determine any value. Then one always comes up against the phenomenon of fatigue which manifests itself in different degrees depending on the strength and type of stimulus as well as the type of individual tested. The fatigue may be (1) that of attention, (2) psychic or (3) end-organ or physiologic. Finally, one meets with a special situation in the form of parosmia or perverted sense of smell (congenital or acquired) in which, instead of the expected odor, others are experienced (e. g., florid odors appearing like garlic, xylol like benzine, etc.).

The monograph is brought to a conclusion by a discussion of the classifications of odors and the attempts at establishing psychophysical laws of classification. The author suggests grouping these, for the purposes of showing quality interrelations and passages of one into the other, in the form of a prism

(analogous to the optic octahedron). At the triangle at one end of the prism, he places the odors — flowery, putrescent and fruity and at the other end, aromatic (spicy), empyreumatic (burned) and resinous.

The presentation of the methods of the experimental approach, as well as of the sources of error, is thus seen to be masterfully accomplished. The purely theoretical considerations, however, as well as the presentation of modern concepts on the subject seem to leave some room for discussion. The author, after establishing the psychologic "complex" value of a perception, proposes to consider such a complex in the light of a chemical compound which can be reanalyzed into its components. These components he appears to be able partially to isolate and measure, regarding the complex value as something to be considered, no doubt, but as a quality only. One would wonder whether it were not more consistent to regard the whole complex as an indivisible unit, and its so-called components as qualities.

In the discussion of Gestalt and complex conceptions (p. 803), one is surprised to see no mention of, or reference to, the whole literature of the modern Gestalt psychology (Köhler, Wertheimer, Goldstein, etc.) even though no such work has been done on the olfactory sense. Then again, in the discussion of sensibilitization and interrelations of different sense organs, the work of the Russian school on the conditioned reflex is not taken up.

The principal object of the work, however, is that of presenting the methods of psychologic investigation. This is carried out splendidly and, as such, is highly recommended to students in this field.

DER HEUTIGE STAND DER BEHANDLUNG DER PROGRESSIVEN PARALYSE (THE PRESENT STATUS OF THE TREATMENT OF GENERAL PARALYSIS). By LADISLAUS BENEDEK. Price, 12 marks. Pp. 204. Berlin: S. Karger, 1926.

This is an excellent treatise on the problems of general paralysis. It is, of course, possible to offer criticisms; for instance, insufficient attention is given to American work, although the author does rarely refer to some American worker. One might criticize the handling of the subject, as too much weight at times is given to minor details and matters of considerable importance are treated lightly. The main defect, which is undoubtedly incidental to the small amount of attention given to American work, is the absence of any mention of tryparsamide. All these criticisms and others that might be made can be easily avoided in future editions, which this treatise merits. It is written by a man with long experience in the treatment of general paralysis by many methods, so he is able to discuss the subject from the standpoint of one who has worked as well as one who has read and thought.

Even though the bibliography does lack the names of important American contributors, it is very rich indeed. The reviewer feels justified in saying that this book should be studied by every worker in the field of neurosyphilis. Certainly, every one who is treating or advising in the treatment of the cases of general paralysis should be familiar with its contents, and it is the reviewer's belief that nowhere else will one find under one cover so good a review of the whole problem.

The author starts with the discussion of the fundamentals of the problem of general paralysis, discussing the histopathology, the problems in etiology, and then the parasitology and pathogenesis. In these relatively short sections there is a wealth of material often only lightly mentioned but, on the other hand, sufficient to give one many ideas. Considerable emphasis is put on the discussion of problems of immunity. Matters such as the reaction of the

ectodermal tissue, the meaning of luetin reactions and the significance of hyperallergia are discussed. This may give some idea of the background which precedes the major part of the work dealing with the effect of treatment.

The high point in the treatment of general paralysis as seen by the author is the use of infections, as malaria and relapsing fever. He gives, however, a good summary of the work of Wagner and Pilcz which preceded the malarial method. This includes the treatment with tuberculin and mercury and certain other fever-producing substances as the vaccines. There is also a review of the production of fever by drugs such as sodium nucleinate, protein and milk injections. Benedek not only reviews the literature of this work but is able also to tell of his own experiences, and one gathers that he believes that these methods really produced satisfactory results on the whole, though not nearly as satisfactory as treatment with malaria. There is an excellent review of the literature, particularly that of the workers in central Europe.

The summing up of the results of malarial treatment is very encouraging. The summary would seem to show that between 20 and 30 per cent of the patients with general paralysis treated by malaria showed excellent remissions which enabled them to return to their former occupations; up to 70 or 80 per cent showed considerable improvement, although not as marked as that of the first group.

In attempting to evaluate these results and get something of their significance it is necessary to compare the treated patients with the untreated having spontaneous remissions, and a very good review of the studies from central Europe concerning spontaneous remissions is given. On the whole, the story of the spontaneous remissions is that they are much less frequent than those obtained in treated patients and of much shorter duration. The average patient with a spontaneous remission continues in apparently good condition for about six months, while only 10 per cent have a remission lasting two years or more. Benedek goes into considerable detail regarding the technical side of malarial treatment, considering the incubation periods, the types of reaction, etc. This is helpful to those using or intending to use the method. He concludes that the malarial method is not nearly as satisfactory in cases of tabes as in cases of general paralysis.

The author goes into some detail on the matter of combining antisyphilitic treatment with malarial therapy. He quotes Kirschbaum as thinking that the combination has no advantage over malaria alone; in fact, he rather felt the reverse to be the case, and in this view Nonne and Weygandt concur. Dattner goes so far as to say that arsphenamine is contraindicated after malaria because the latter affects the liver, which loses its detoxicating influence; following arsphenamine treatment the brain is likely to be affected and the patient to develop paranoid states. He also thinks that malaria may affect the suprarenal bodies, and thus the use of arsphenamine may prove fatal. It is impossible in a short review to touch on the many technical problems that are discussed in this relation, but they are extremely worth while and merit careful study.

A small amount of space is given to the relapsing fever method in treating general paralysis, as used by Plaut and Steiner. The conclusions are that the results are quite similar to those obtained with malaria. The effect of this treatment on the serology is discussed in an interesting fashion. Apparently, Wagner has little interest in the effect on the serology because he feels that in cases in which the serology has been favorably influenced the patients may not do particularly well clinically, while, on the other hand, they may make

excellent clinical improvements without doing well from the serologic standpoint. It has been shown, however, that in many of the cases serologic improvement begins from six months to a year after the treatment.

As to the theoretical considerations of why the malarial or associated methods should have a beneficial effect, Benedek finds no entirely satisfactory answer. The discussion of this is worth reading in detail. He calls attention to the fact that fever itself is hardly a sufficient explanation, nor is there any clear-cut evidence of a real immunity effect. If he has any belief, it is that there is a change in the "Abwehrreaktion" in which the cells of the reticulo-endothelial structures play the chief rôle.

A fairly detailed discussion of the specific treatment of early syphilis in relation to the later development of neurosyphilis is given. There are also discussions of intrathecal methods of treatment including the double puncture methods of Generich. Benedek also calls attention to the tendency of some of the Viennese clinics to treat early syphilis by a course of arsphenamine followed by a course of malaria, with the idea of prevention of later central nervous system involvement.

Finally, there is a brief chapter on the use of stovarsol, a drug prepared by Levaditi and Navarro in 1922, which, it is claimed, when taken by mouth prevents the development of syphilis.

It is the reviewer's hope that this brief summary gives some idea of the richness of the material contained in this book. Any one giving careful attention to it cannot help being impressed by the fact that general paralysis offers innumerable problems and that we are only at the beginning of our understanding of the etiology, parasitology, bodily responses, methods of treatment, and interpretation of clinical manifestations. One is certainly impressed with the fact that to understand the numerous problems of general paralysis one must have considerable knowledge of modern biology and medical sciences.

PSYCHIC RESIDUALS OF EPIDEMIC ENCEPHALITIS IN CHILDREN, ESPECIALLY THE FURTHER DEVELOPMENT OF THESE CASES. By RUDOLPH THIELE. Pp. 100. Berlin: S. Karger, 1926.

Thiele reports the observation over several years of twelve cases of epidemic encephalitis in children who showed psychic symptoms following the disease. He believes that the psychosis is well defined and easily differentiated from most other psychoses. Very full case histories are given, from which the conclusions are drawn.

Restlessness is an outstanding feature in all cases. The pseudovoluntary movements are so irregular that in some cases they resemble choreiform activity rather than psychomotor hyperactivity. This activity seems to be in the nature of a compulsion that does not bring contentment to the patients, of which they are aware, and dislike, but of which they feel the urgent necessity. Most of the action is without motive, such as the impulse to pick up everything and immediately put it down. There does not seem to be a tendency to steal. Probably this is on account of the fluctuating and very superficial interest that is manifested by these patients. Respiratory disturbances, which are fully described, come in crises and sometimes mark the apex of the motor impulsion. The activity is especially marked at night and is sometimes a great distress both to the patients and to those surrounding them. Sometimes the children will get out of bed without warning and strike sleeping patients, immediately climbing back into bed. During the attacks, the patient's consciousness is never disturbed. Even in the height of the agitation they respond relevantly

and promptly, but their attention is easily distracted. Hallucinations are lacking, and orientation is preserved. Emotionally, these patients are usually indifferent but may sometimes show a fear reaction. Memory for the attacks is usually good. Difficulty in concentration is one of the most marked features on the intellectual side, although intellectual defects and memory defects are seldom present. Sometimes there is almost a flight of ideas, but this is probably apparent rather than real, for every thought is immediately blurted out without regard to future consequences. This is another indication of the compulsion.

Mild euphoria and mild depression are sometimes present. Patients are irritable and often break forth into outbursts of anger that render them antisocial, although criminal acts are rather infrequent. Patients show impulsive badness sometimes amounting to viciousness and may be labeled born criminals by teachers who do not understand them, but they are usually sorry for their misdeeds and offer to make amends. They have the feeling that they cannot control themselves. In spite of their great restlessness, they complain of ennui. Sometimes they can predict their outbursts, as one patient, who said, "Look out, doctor, I am not to be spoken to today." They realize that there is "something" within them that causes these outbursts over which they have no control. Some children will blame it on the devil. An interesting autographic observation is given by one patient.

The author doubts whether this alteration constitutes a real character change, since these patients realize that the acts are foolish and that they cannot control themselves, and since they regret their badness. Except for this, their behavior would be very much like that of a constitutional psychopath. The psychic manifestations may be due to actual pathogenic action of the disease on the cerebral tissue, or to the basic character freed from control or to the regressive tendency of their character whereby the operations of a childish mentality are set back to a more infantile level. Occasionally these patients, particularly the girls, manifested eroticism and solicited the attention of strangers. Hysterical reactions were fairly frequent.

In the years that followed the onset of the psychosis, notable improvement was registered in a large percentage of the cases; so much so that some of the patients recommenced their schooling or took up some different occupation. There were still irritabilities and restlessness noted in many instances. Disturbances of sleep were common even long after the onset of the disease, and were about the last to disappear. Dementia was rare, but occurred most markedly in the youngest patients. The onset of the amyostatic syndrome was "signum mali ominis." The psychic symptoms might come on a long time after the acute attack.

The literature on this condition is reviewed, but the author has apparently made a real contribution to the later course of these cases.

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